ANALYSIS OF CARDIAC MANIFESTATIONS IN HIV

Dissertation submitted in partial fulfillment of requirements for

M.D. DEGREE IN GENERAL MEDICINE

BRANCH 1

of

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY,

CHENNAI, INDIA.

MADRAS MEDICAL COLLEGE,

CHENNAI – 600 003.

APRIL 2011

DECLARATION

I solemnly declare that this dissertation entitled **"ANALYSIS OF CARDIAC MANIFESTATIONS IN HIV"** was done by me at Madras Medical College & Government General Hospital, Chennai – 3, during 2010 under the guidance and supervision of Prof. C. Rajendiran, M.D. This dissertation is submitted to the TamilNadu Dr. M. G. R. Medical University towards the partial fulfillment of requirements for the award of M. D. Degree in General Medicine (Branch 1).

Place: Chennai-3 Candidate's Signature

Date:

ACKNOWLEDGEMENT

At the outset I thank **Prof. J. Mohanasundaram M.D.,** Dean, Madras Medical College and Government General Hospital, Chennai-3 for permitting me to use hospital data for the study.

I am grateful to **Prof. C. Rajendiran, M. D.,** Director and Head of Department, Institute of Internal Medicine, Madras Medical College and Government General Hospital, Chennai-3 for his support.

I would also like to thank **Prof. S. Ragunanthanan,** ART center, Madras Medical College and Government General Hospital, Chennai-3 for his support.

I also express my gratitude to **Assistant Prof. Dr. R. Muthuselvan, M.D.** and **Dr. S. Baskar, M.D.,** Madras Medical College and Government General Hospital, Chennai-3.

My sincere thanks to all the patients who participated in this study.

Lastly, I thank all my professional colleagues for their support and valuable criticisms.

CERTIFICATE

This is to certify that the dissertation entitled **"ANALYSIS OF CARDIAC MANIFESTATIONS IN HIV"** is a bonafide work done by Dr. V.Nalini at Madras Medical College, Chennai in partial fulfillment of the university rules and regulations for award of M.D., Degree in General Medicine (Branch 1) under my guidance and supervision during the academic year 2010.

Prof.C.Rajendiran, M.D
Director and Professor,
Institute of Internal Medicine,
Madras Medical College &
Govt. General Hospital,
Chennai - 3.

Prof.J.Mohanasundaram,M.D, DNB, Phd,
The Dean,
Madras Medical College&
Govt. General Hospital,
Chennai - 3.

CONTENTS

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) is characterized by an acquired, profound, irreversible immunosuppression that predisposes the patient to multiple opportunistic infections, malignant neoplasms, and a progressive dysfunction of multiple organ systems. The heart and great vessels are not the sites most frequently affected by opportunistic infections and neoplastic processes in patients with acquired immune deficiency syndrome (AIDS). However, cardiovascular complications occur in a significant number of such patients and are the immediate cause of death in some.

The prevalence of cardiac involvement in AIDS patients has been reported to range between 28% and 73%[1]. Recent advances in the knowledge about human immunodeficiency virus (HIV) replication and the treatment of HIV infection have improved survival in HIV patients[2-4]. Because of the longer survival in HIV patients, the more manifestations of late-stage HIV infection will be seen, including HIV related cardiac diseases. As patients with HIV infection are living

longer, they are at risk of developing chronic diseases, including coronary atherosclerosis too. The spectrum of cardiovascular complications of AIDS that may be depicted at imaging includes

a) Dilated cardiomyopathy
b) Pericardial effusion
c) Human immunodeficiency virus–associated pulmonary hypertension
d) Endocarditis
e) Thrombosis
f) Embolism
g) Vasculitis
h) Aneurysm
i) Coronary artery disease
j) Cardiac involvement in AIDS-related tumors.

In this article, we review the pathophysiologic and imaging manifestations of HIV and AIDS related cardiovascular complications.

OBJECTIVE

To identify the incidence of cardiovascular diseases in various stages of AIDS by clinical profile, electrocardiogram and echocardiography.

DESIGN

Prospective, clinical, and echocardiographic study.

SELECTION OF PATIENTS

INCLUSION CRITERIA:

150 consecutive HIV-infected patients attending the Institute of Internal Medicine both as inpatients and outpatients who were positive for HIV antibodies were included in this prospective study irrespective of their duration of their illness and ART status.

EXCLUSION CRITERIA:

Patients with evidence of heart disease previous to the diagnosis of their HIV infection were specifically excluded.

STUDY DURATION: JAN 2009 TO JUNE 2010

SAMPLE SIZE: 150 patients

MATERIALS AND METHODS

150 consecutive HIV infected patients attending the Institute of Internal Medicine both as inpatients and outpatients who were positive for HIV antibodies were selected.

All the patients were given a thorough clinical examination that included a patient history, a physical examination. Routine lab investigations like RFT, LFT, CHG, fasting blood sugar and CD4 count were taken. ECG and two-dimensional echocardiography were taken and cardiac findings were analyzed statistically.

REVIEW OF LITERATURE

Cardiac involvement in AIDS was first reported in 1983 by Autranet al[5]. They described a 24 years old Haitian woman with multiple complications of AIDS who, at postmortem, had Kaposi's sarcoma (KS) involving the entire anterior cardiac wall without pericardial effusion. Since the time of that report, the understanding and the documentation of AIDS related complications have evolved considerably and reports of lesions related to the heart have increased.

It is now apparent that HIV, secondary opportunistic infections, and the medical treatment itself of HIV disease can all affect the heart[6-8]. The spectrum of abnormalities ranges from clinically silent lesions detected on postmortem examination to clinically potentially fatal diseases, such as pericardial tamponade.

PERICARDIAL EFFUSION

The spectrum of pericardial disease in HIV infected patients is wide. It ranges from asymptomatic effusions detected on echocardiography to potentially fatal tamponade and constrictive pericardial disease [9-12].

ETIOLOGY

Acute and chronic pericarditis can also occur. Pericardial disease in HIV-infected patients may have infectious, noninfectious, or neoplastic etiologies. Infectious causes of pericardial disease in HIV/AIDS include viruses, bacteria, and fungi. *Cytomegalovirus (CMV)* is a frequent pathogen in patients with AIDS and has been reported to cause cardiac tamponade in a patient with cryptococcal meningitis[13]. *Herpes simplex virus* is also reported as a cause of pericarditis in AIDS patients[14-15]. It remains unclear whether HIV itself can independently cause pericarditis[16]. Bacterial pericarditis can be caused by *Staphylococcus aureus*[17-18] and *Klebsiella pneumoniae*[19], as well as *Nocardiaasteroides*, *Mycobacterium tuberculosis*[20-21], and *Mycobacterium avium* complex in HIV-infected patients with Disseminated infections. Fungal pericarditis secondary to *Cryptococcus neoformans*[22-23] has also been reported. Cardiac lymphoma in HIV patients has been described in many reports[24-25]. It can be primary or secondary and can involve the pericardium. Kaposi Sarcoma has also been described and can affect the pericardium as well as the epicardium and can rarely cause tamponade. Nonetheless, many pericardial effusions appear to be idiopathic[26].

CLINICAL FEATURES

The clinical picture of pericardial disease can be silent or may present with chest discomfort, dyspnoea and fever. Physical examination may reveal tachycardia, tachypnoea, and friction rub. Cardiac tamponade usually manifests with tachypnoea, hypotension with pulsus paradoxus, elevated jugular venous pressure and distant heart sounds. "Low-pressure tamponade" is a phenomenon that is sometimes observed in AIDS patients who are severely dehydrated or cachectic; the severe volume depletion may cause reduced right ventricular filling pressure, and a minimal pericardial effusion may cause a hemodynamically significant tamponade. In this setting, elevation of jugular venous pressure or pulsus paradoxus may be absent[27]. The cardiac dysfunction may be easily obscured or unrecognized because the clinical picture of AIDS may be so overwhelmingly dominated by the impact of opportunistic infections.

DIAGNOSIS

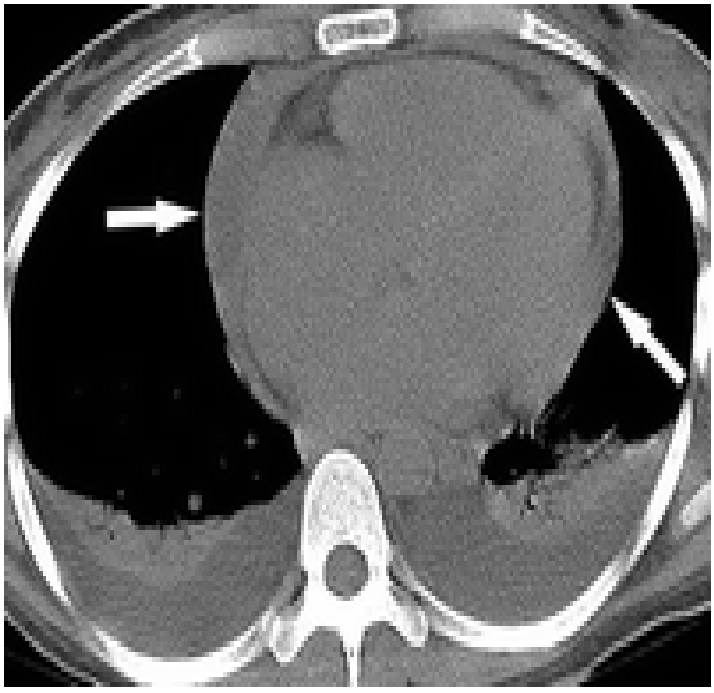
The patient's chest radiograph may or may not show an enlarged cardiac silhouette with classic "water-bottle" appearance depending on the size of effusion. Electrocardiography (ECG) may demonstrate ST-T

wave changes indicative of pericarditis. As pericardial effusion increases, low QRS voltage and electrical alternans may appear. Echocardiography is an excellent diagnostic tool for detecting and monitoring pericardial effusion and tamponade[28-30].

TREATMENT

When clinically significant pericardial effusion is encountered in an HIV infected patient, the differential diagnosis should first exclude the non HIV related causes, such as renal failure, trauma, radiation effect, drug toxicity, connective tissue diseases, and hypothyroidism. Small asymptomatic effusions can be observed clinically and with serial echocardiograms. Nonsteroidal anti-inflammatory agents (NSAIDs) may provide some relief. Corticosteroid therapy should generally be avoided because of the likelihood of concurrent infection, which may be exacerbated by steroid therapy. Diagnostic pericardiocentesis or biopsy may be indicated for microbiologic and cytologic studies when an infectious etiology is highly suspected or when presumed infectious pericarditis fails to respond to empiric antibiotics. A diligent attempt must be made to establish an etiologic agent. This is critical because some of these pericardial infections (e.g., tuberculosis) are amenable to specific therapy, even in immunocompromised individuals.

Cardiac tamponade requires an urgent intervention with pericardiocentesis. Removal of a small amount of fluid often produces immediate hemodynamic benefit, but complete drainage with a catheter is preferable. Draining pericardiotomy or pericardiectomy may be appropriate for recurring effusion especially in neoplastic disease and chronic pericarditis. Additional therapy is determined by the extent of the response to initial management.



Pericardial fluid in a 32 years old woman with AIDS and a history of recurrent pneumonia. Unenhanced axial computed tomographic (CT) scan demonstrates a medium-sized pericardial effusion (arrows), as well as bilateral pleural fluid collections and slight dilatation of the heart.

MYOCARDIAL DISEASE

Four forms of myocardial disease have been described in patients with HIV. The first is myocarditis, which may result from invasive opportunistic infections or lymphocytic infiltration. Non-inflammatory myocardial necrosis has also been described. The third form is dilated cardiomyopathy, and the fourth form is infiltrative myocardial disease caused by neoplasia such as Kaposi Sarcoma or high-grade HIV-related lymphoma.

DILATED CARDIOMYOPATHY

Dilated cardiomyopathy in the setting of HIV/AIDS reflects severe cardiac dysfunction. Biventricular or four-chamber dilation is the characteristic finding. It may be either primary[31, 32] or secondary as a result of chronic active myocarditis, cardiotoxins, hypersensitivity reactions[33] , and certain dietary deficiencies such as thiamine (vitamin B_1)[33,34] and selenium[26].Myocardial damage related to hypersensitivity reaction and cardiac specific autoimmunity leading to myocarditis, cardiomyopathy, or both has been suggested in some published studies[33-41]. Alteration of T-helper cell function, as seen in patients with AIDS, may result in uncontrolled hypergammaglobinemia and high

concentration of serum immune complexes that have the potential for causing local and systemic inflammatory lesions in the major organs of body including the cardiac muscle itself[33]. Other studies postulated that the HIV gene alters the surface of the cardiac muscle fiber inducing cell-surface immunogenic proteins and leads to the formation of circulating cardiac auto antibodies, that can trigger a progressively destructive autoimmune reaction. Such heart specific auto antibodies have been demonstrated in some HIV-positive patients with cardiomyopathy[34-41]. Whether such immunologic changes can be attributed to only HIV infection remains unclear, and further research may still be needed in this area.

Nutritional deficiencies and severe wasting syndrome are common problems in terminally-ill patients with AIDS[31, 42]. Vitamin deficiency, particularly vitamin B1, may occur in such situations and may play a role, although secondary, in AIDS-associated dilated cardiomyopathy[26,33]. Selenium deficiency and its association with cardiomyopathy in malnourished pediatric AIDS patients have also been described; selenium supplementation in this subgroup of AIDS patients has been shown to improve cardiac dysfunction[42-44]. It is still believed, however, that such nutritional deficiencies may have a secondary role in

the pathogenesis of cardiomyopathy, and additional studies may be required to determine the effect of various dietary supplements on the reversibility of cardiac dysfunction in these patients.

CLINICAL FEATURES

Clinical manifestations of myocardial lesions depend on the severity of the inflammatory process and the location of the lesion. Subclinical conditions are not uncommon. When symptomatic, the associated symptoms include chest pain, dyspnea, fatigue and peripheral edema. Palpitations, dizziness and syncope may indicate dysrhythmia. Physical examination may reveal signs of biventricular failure with valvular dysfunction.

DIAGNOSIS

Chest radiography shows cardiomegaly and pulmonary vascular redistribution. ECG may reveal various types of dysrhythmias and even a pseudoinfarct pattern. Echocardiography is helpful in diagnosing cardiomyopathies and to a lesser extent, neoplastic infiltrations, which are more likely to be detected on CT or MR. Isolation and identification of specific etiologic agents are critically important especially in life-threatening conditions. The necessity of performing myocardial biopsy

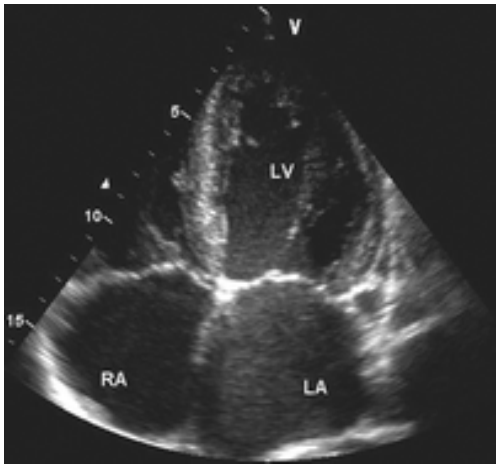
remains controversial, and the risk associated with this procedure may be significant. It may be diagnostically helpful in rare situations, particularly in the setting of isolated cardiac tumors and in patients with extensive cardiac damage and poor prognosis without a known identifiable cause. However, it may be of limited sensitivity, especially in patchy lesions and only rarely useful in the management of the patient. It is a procedure that has considerable risks, which should always be weighed against the expected benefit.

TREATMENT

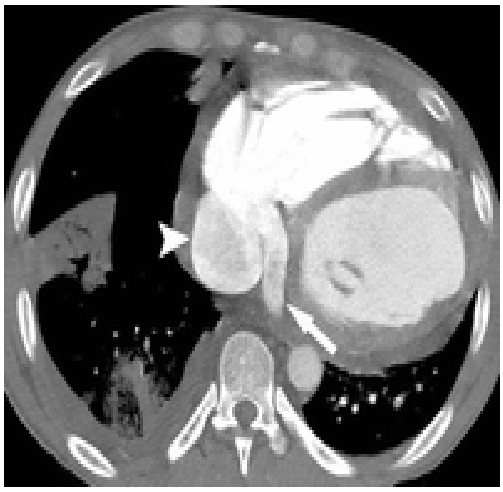
HIV-related cardiomyopathy and associated congestive heart failure (CHF) may respond to standard management, including inotropic agents, diuretics and vasodilators. Antidysrhythmics should be used where indicated. The spectrum of dysrhythmia in patients with myocarditis may be wide. It includes various atrial and ventricular rhythm disturbances as well as various degrees of heart block. The antidysrhythmic agents to be used depend on the type of dysrhythmia, its hemodynamic consequences, and the associated adverse effects that may accompany the use of such agents, for example, negative inotropic effect[45].

Anticoagulants may also be used in certain conditions, such as atrial fibrillation or when the left ventricular systolic function is markedly reduced. However, the benefit of anticoagulation should always be balanced against the risk of having a bleeding complication especially in AIDS patients; not only because myocarditis may be associated with pericardial disease, which may lead to intra pericardial hemorrhage, but also because AIDS patients are at higher risk of having vasculopathy with aneurysmal formation that may lead to intra cerebral bleeding from mycotic aneurysms[9].

Dilated cardiomyopathy in a 38 years old man with AIDS. Radiograph shows an enlarged cardiac silhouette with left ventricular enlargement



Apical four-chamber echocardiographic view shows marked enlargement of all four chambers, as well as spontaneous echo contrast ("smoke") in the left-sided chambers because of a very low ejection fraction and sluggish flow.



Contrast-enhanced axial CT scan demonstrates significantly enlarged right and left ventricles and a dilated inferior vena cava (arrowhead) and coronary sinus (arrow), as well as pericardial fluid, right-sided pleural effusion, and parenchymal consolidation in the lower lobe of the right lung.

MYOCARDITIS

Myocarditis in HIV patients may play a role in the development of ventricular dysfunction.The autopsy incidence of myocarditis was approximately one third of all AIDS patients. A specific cause was found in less than 20% of these patients. Common pathogens in AIDS myocarditis include *Toxoplasma gondii*, *Mycobacterium tuberculosis* and *Cryptococcus neoformans*. Other infectious organisms have been reported to include *Myocobacterium avium-intracellulare complex*, *Aspergillus fumigatus*, *Candida albicans*, *Histoplasma capsulatum*, *Coccidioides immitis*, *cytomegalovirus,* and *herpes Simplex 1 and 2*[26]. Recent data suggested that HIV alone can cause myocarditis. Either HIV or its proteins(p17, p24, and gp120/160) have been found in the heart specimens of patients with AIDS with or without cardiac diseases by culture, in situ deoxyribonucleic acid hybridization, and by Southernblot tests[46-48].

Superantigen plays an important role in the pathogenesis of many diseases by forming a trimolecular complex with major histocompatibility complex class II molecule on the antigen-presenting cells and the Vβ-specific region on the T-lymphocyte receptor[49]. The binding results in a massive stimulation of the T lymphocyte. After the

binding of HIV regulatory protein (Nef) with major histocompatibility complex class II on antigen-presenting cells, the T lymphocytes become activated. The activation of T lymphocyte stimulates the proliferation and release of cytokines such as interferon γ and interleukin 2. Therefore, the viral load in the heart will increase from creating a cellular reservoir for HIV. T lymphocyte depletion may be caused by apoptosis, anergy or both. Proliferation of the B cell may result in hypergammaglobulinemia. Autoimmune response may occur as a result of B cell differentiation into immunoglobulin-secreting cells and activation of the T lymphocyte.

Lymphocytic myocarditis was found in 37 of 71 patients who died of AIDS[50]. There were 3 types of histological features: lymphocytic infiltrate with necrosis of the myocardial fibers,lymphocytic infiltrate without necrosis of the myocardial fibers[51], and focal and mild myocarditis with a mononuclear infiltrate[50].

PULMONARY HYPERTENSION

It is more common in male and young patients (mean age, 32 years). The common risk factors are intravenous drug use, homosexual contacts and hemophilia. The major symptom of this conditionis

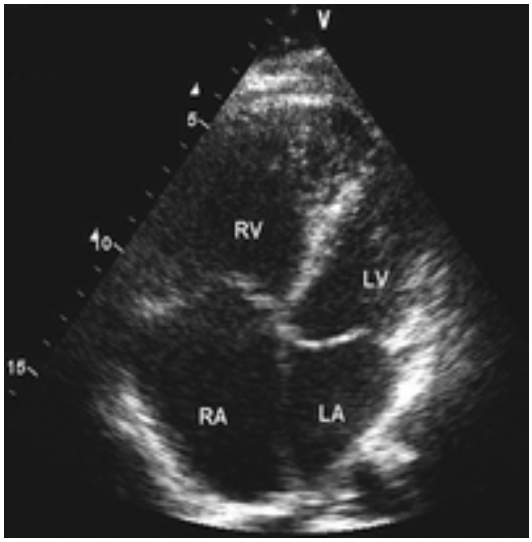
dyspnea[52]. There was no correlation between both a history of opportunistic infections or CD4 cell count and the development of pulmonary hypertension. The mean pulmonary artery systolic pressure was 68 mm Hg. The major causes of death were right sided heart failure and respiratory failure.

The exact mechanism by which pulmonary arterial hypertension develops is not clear, although various causal factors have been proposed, including genetic predisposition, peri-vascular inflammation, increased expression of vascular endothelial growth factor A (a potent inducer of the growth of endothelial cells, which increases vascular permeability) in T cells, increased expression of intrapulmonary platelet-derived growth factor, and HIV-induced release of active endothelial cellular mediators[52]. Glycoprotein 120, an *HIV-1* envelope glycoprotein, has been found to stimulate the production of endothelin 1 and tissue necrosis factor α and too chronically increase the expression of endothelin 1, a pulmonary vasoconstrictor[52].

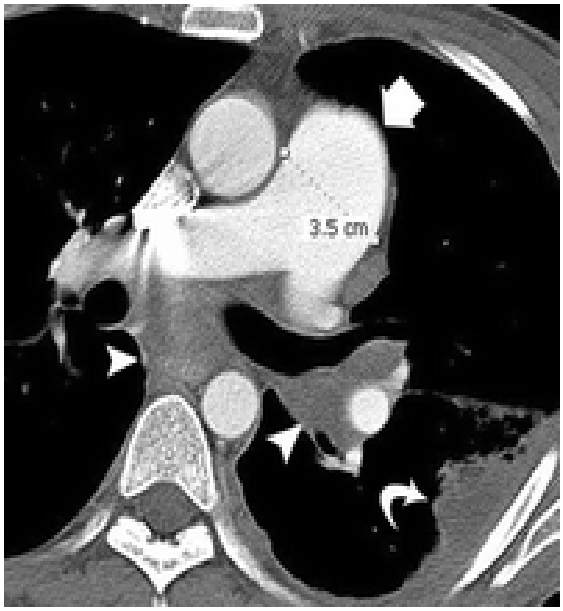
In most of cases, pulmonary arterial hypertension is due solely to HIV infection. Pulmonary arterial hypertension also has been attributed to recurrent pulmonary infection with secondary pulmonary inflammation and fibrosis, to intravenous drug abuse, and to cirrhosis.

Findings at histopathologic analysis of lung tissue from patients with the condition are similar to those of primary pulmonary hypertension. The most common finding is plexogenic pulmonary arteriopathy[52,53]. Less common pathologic findings include medial hypertrophy and intimal fibrosis without plexiform lesions, pulmonary veno-occlusive disease and thrombotic pulmonary arteriopathy[52].

The imaging findings in these patients also are similar to those observed in primary pulmonary arterial hypertension. They include enlargement of the pulmonary artery trunk, prominence of the central pulmonary arteries, tapering of peripheral vessels, and an enlarged cardiac silhouette secondary to right ventricular and right atrial dilatation (72% of patients). Abnormalities in cardiac morphologic features may be visualized with contrast-enhanced CT. These include right atrial and ventricular dilatation, often with reversal of the curvature of the interatrial and interventricular septa followed by tricuspid regurgitation and paradoxical septal motion[52]. Other echocardiographic findings are right ventricular wall hypertrophy, septal impingement on the left ventricle and pulmonary valve insufficiency[54].



Apical four-chamber view of the heart depicts marked enlargement of the right-sided chambers, with a right ventricular diameter almost twice the diameter of the left ventricle



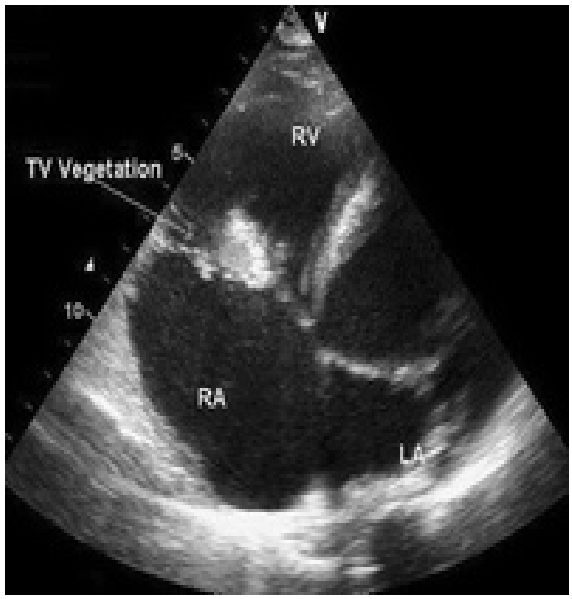
Contrast-enhanced CT scan depicts the abnormal prominence of the main trunk of the pulmonary artery (35 mm) (straight arrow).

ENDOCARDITIS

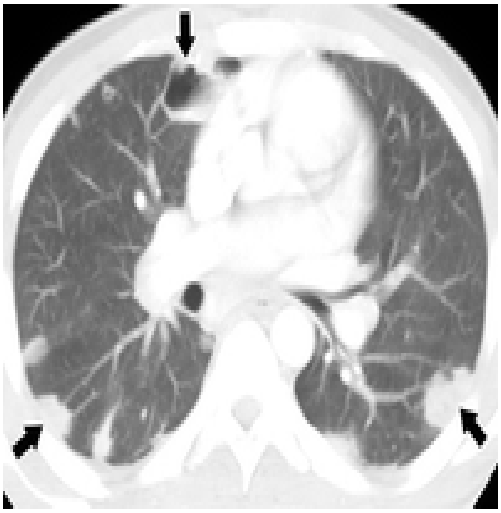
Marantic endocarditis or nonbacterial thrombotic endocarditis is characterized by friable, fibrinous clumps of platelets and red blood cells adherent to the cardiac valves without an inflammatory reaction. It is estimated that this condition occurs in 3% to5% of AIDS patients[55]. Marantic endocarditis is known to be associated with malignant neoplasms, hypercoagulable states, and chronic wasting disease[56-58]. Mitral and aortic valves are commonly involved in HIV-negative patients[57], but the tricuspid valve is usually involved in AIDS patients[50]. Systemic embolism can occur in upto 42% of patients, but most of these events are clinically silent. Embolization can involve the brain, lung, spleen, kidney and coronary arteries[59]. Systemic embolization from marantic endocarditis is a rare cause of death in AIDS patients.

Infective endocarditis in patients with AIDS usually occurs in parenteral drug users. Human immunodeficiency virus infection may increase the risk of infective endocarditis among intravenous drug users. The major causes of infective endocarditis in HIV patients are *Staphylococcus aureus* and *Streptococcus viridans*. Other unusual organisms described as case reports were *Salmonella*, *Aspergillus fumigatus* and *Pseudallescheriaboydii*. The tricuspid valve is the most

commonly affected valve. The affected patients usually present with fever, sweats, weight loss and coexisting pneumonia and / or meningitis. The presentation and survival of infective endocarditis in patients with and without HIV infection are generally not different; however, in the late stage of HIV-infected patients, significant increased mortality from infective endocarditis has been reported compared with asymptomatic HIV patients[60].



Right parasternal long-axis four-chamber echocardiographic view shows marked right atrial enlargement and a large area of echogenicity adjacent to the tricuspid valve.



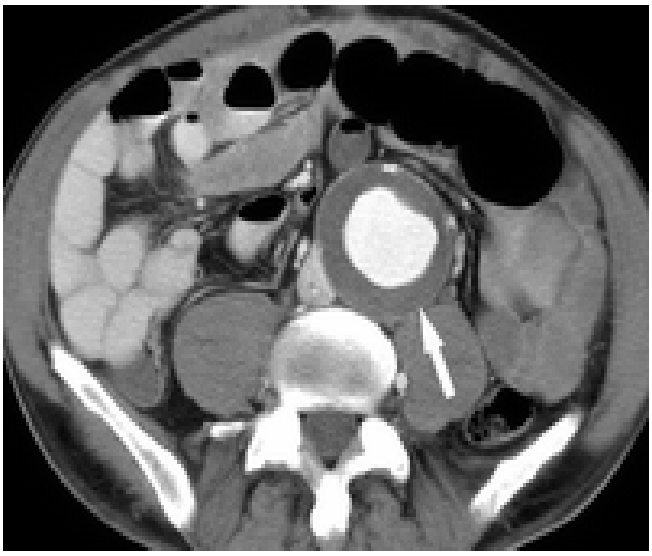
Axial chest CT scans obtained with lung window settings demonstrate cavitary and noncavitary nodules (arrows in **a**) in both lungs.

VASCULITIS

A range of inflammatory vascular diseases, both infective and noninfective, may occur in the setting of HIV. These include polyarteritis nodosa, Henoch-Schönlein purpura and drug-induced hypersensitivity vasculitis[61]. Features similar to those in Kawasaki syndrome, coronary arteritis and Takayasu arteritis also have been described[62].

Large-vessel disease may be aneurysmal or occlusive. Aneurysms may be single or multiple and may affect vessels such as the aorta or the common carotid, common iliac, femoral, or popliteal arteries[63].

Occlusive disease has been reported in Africa in young HIV-positive patients and is less common than aneurysmal dilatation[63,64]. In both processes, the main histopathologic features are found in the adventitia, with leukocytoclastic vasculitis of the vasa vasorum and periadventitial vessels, chronic inflammation and fibrosis. Accelerated atherosclerosis also was found responsible for aortic aneurysm in a young man who had undergone several years of HAART for AIDS [65].



Contrast-enhanced CT scan of the abdomen shows the dilated aorta with a mural thrombus (arrow).

THROMBOSIS AND EMBOLISM:

HIV-infected patients tend to develop coagulation abnormalities due to increased levels of fibrinogen, d-dimer, plasminogen activator inhibitor-1, and tissue-type plasminogen activator antigen. A deficiency of protein S also has been identified. These hematologic abnormalities are associated with both arterial and venous thrombosis, especially in patients who are undergoing therapy with protease inhibitors [66,67]. In a series of HIV-positive patients with venous or arterial thrombosis, deep vein involvement, most commonly in a lower extremity, and secondary pulmonary emboli accounted for 66% of all thrombotic events. Persistent antiphospholipid antibodies were the most common abnormal serologic finding[68]. An association between cigarette smoking and spontaneous thrombosis in HIV patients has been mentioned [67] and was reported to affect 77% of the patients in one series[68.]

Contrast-enhanced chest CT scans show bilateral pulmonary embolisms (arrows).

CORONARY ARTERY DISEASE

Antiretroviral medications have dramatically reduced the overall death rate among patients with the human immunodeficiency virus (HIV), but those same patients may now face an increased risk for cardiovascular disease (CVD) [69].

Many patients infected with HIV have cholesterol abnormalities which may be side effects from the antiretroviral medications, effects of the virus, or both. Also common in this population are traditional risk factors for cardiovascular disease such as insulin resistance, diabetes, the tendency to carry excess weight in the mid-section, kidney abnormalities and more, which might be influenced by the HIV drug regimen or the virus itself. In addition, there is a high prevalence of smoking in HIV-infected individuals.

Scientists and healthcare providers convened in June 2007 for the State of the Science Conference: Initiative to Decrease Cardiovascular Risk and Increase Quality of Care for Patients Living with HIV/AIDS, a joint effort of the American Heart Association and the American Academy of HIV Medicine.

Among the group's key findings:

- ♣ The risk for heart attack is 70 percent to 80 percent higher among people with HIV compared to those who do not have HIV, although the absolute risk still remains low for younger patients.

- ♣ Having HIV is associated with increases in two important risk factors for heart disease:

  - low levels of HDL cholesterol (high-density lipoproteins or "good cholesterol) and
  - elevated levels of triglycerides.

- ♣ Large studies suggest that the cardiovascular disease increase among HIV- Infected patients is associated with specific metabolic abnormalities linked to antiretroviral therapy, including diabetes, as well as traditional risk factors such as smoking.

- ♣ While it's easy to assume that long-term ART use could further increase cardiovascular risk for these patients, the research suggests that uninterrupted ART treatment may reduce cardiovascular risk.

- ♣ It is important to stratify CVD risk in HIV-infected patients. Clinicians can use existing general risk stratification algorithms, such as the Framingham Risk Score, to measure HIV patients' risk for heart disease, as this algorithm performs reasonably well in the HIV population although it does not incorporate specific HIV factors. Development of well-validated HIV specific risk-prediction equations is an important future research priority.

- ♣ Whether the elevated risk for heart disease is more attributed to the disease, ART or an interaction between the two is not yet clear[69].

HIV AND DIABETES

Human immunodeficiency virus (HIV) patients with heart disease or diabetes have a higher risk for subsequent coronary events than HIV patients without heart disease or diabetes, according to a report in *Circulation: Journal of the American Heart Association*[69].

Researchers investigated the risk of a new episode of coronary heart disease from a database of more than 33,000 HIV patients in Europe, Australia and the United States according to their histories of

coronary heart disease and whether they had diabetes or not. They found:

- Six-hundred and ninety-eight HIV patients experienced a coronary heart disease event, such as heart attack, the need for a coronary procedure or death, during the data collection from 1999 to 2007.
- Patients who had previously had a coronary heart disease event were 7.5 times more likely to have a new event compared to those who had not previously had an event.
- Patients with diabetes were 2.4 times more likely to experience a coronary heart disease event than those without diabetes.

An increasing number of HIV patients are being diagnosed with diabetes, which may be a result of high levels of conventional risk factors (such as older age, physical inactivity and obesity). But diabetes may also occur as a side effect of some of the drugs used to treat HIV.

There is a need for targeted interventions, such as regular physical exercise, smoking cessation and use of cholesterol-lowering drugs, to reduce the risk of coronary heart disease in patients with diabetes and also in those with a history of a coronary heart disease in patients with HIV.

HIV AND DYSLIPIDEMIA

HIV has been associated with dyslipidemia independent of antiretroviral therapy[70].Grunfeld et al found that HIV infection was associated with elevated triglyceride levels that worsened with progression of HIV-related disease[71].

Antiretroviral therapy can also contribute to dyslipidemia. Dyslipidemia has been described as being more common and more severe in HIV patients receiving antiretroviral therapy than in patients not on therapy[72-74]. The severity of the dyslipidemia and the typical pattern of the lipid profile differ among and within the classes of antiretroviral agents. Also, dyslipidemia does not develop in everyone who takes these medications, suggesting that host factors play a major role in its development.

Non-nucleoside reverse transcriptase inhibitors have been associated with elevated levels of high-density lipoprotein cholesterol (HDL-C) and total cholesterol[75]. Nucleoside reverse transcriptase inhibitors, on the other hand, are heterogeneous in their lipid effects, which may depend somewhat on interactions with other antiretroviral drugs in the regimen[76,77]. For example, stavudine is often associated

with elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C) and triglyceride levels. Protease inhibitors are generally associated with elevated levels of total cholesterol and triglycerides.

TREATMENT OF CORONARY RISK FACTORS IN HIV PATIENTS

Several measures can help prevent cardiovascular disease, including both lifestyle changes and medical interventions. The National Cholesterol Education Program recommends a Therapeutic Lifestyle Changes (TLC) program that includes a low-fat diet, weight control, and increased physical activity.

Monitoring

All adults should receive periodic blood lipid profiles; a profile done after fasting for 8-12 hours is most (In people with a triglyceride level above 400 mg/dL, LDL should be measured separately rather than derived from an equation.) People starting anti-HIV therapy should have a fasting lipid profile done before treatment begins and every 6-12 months during treatment (or every 1-2 months if baseline lipid levels are high or a person has other risk factors). Blood glucose also should be measured before antiretroviral therapy begins and periodically during

treatment. A family medical history and questionnaire about lifestyle factors is important in helping assess cardiovascular risk.

Smoking cessation

Quitting smoking, or even cutting down, is one of the most important steps people can take to reduce their cardiovascular risk. In some cases nicotine patches or gum, or the antidepressant bupropion may be used to aid smoking cessation.

Heart-healthy diet

A healthy diet includes reduced amounts of fats and cholesterol. The NCEP recommends that 30% or less of total daily calories should come from fat. No more than 10% should come from saturated fat (7% or less if a person is at increased risk for heart disease). The diet should include no more than 300 mg cholesterol daily (200 mg for a person at increased risk for heart disease). Sodium consumption should be no more than 2,400 mg per day. The NCEP also recommends reduced alcohol consumption.

Healthy weight

Weight loss can help lower LDL and triglyceride levels and increase HDL levels. However, if wasting is a problem it may be difficult to obtain adequate calories on a very low fat diet. It is important to consider the relative benefits and risks for each individual. If more calories are needed, it is better to obtain them in the form of unsaturated rather than saturated fat.

Increased physical activity

Exercise can lower LDL and VLDL levels and raise HDL levels; it also can help lower blood pressure. Aerobic exercise works out the heart and lungs. Even moderate exercise -- such as walking 30 minutes per day several times a week can improve cardiovascular health.

Lipid-lowering drugs

Drugs that reduce high blood fat levels may be used if lifestyle modifications are not adequate. Studies in the general population have shown that cholesterol-lowering drugs successfully decrease total and LDL cholesterol levels and reduce the risk of cardiovascular disease. These drugs have not been thoroughly studied in people with HIV, but

early results suggest they are effective. However, some lipid-lowering drugs can interact with antiretroviral drugs and should be used with caution.

- **Statins** are first-line therapy for high cholesterol in HIV negative people; they also may help reduce triglyceride levels. The Adult AIDS Clinical Trial Group (AACTG) Cardiovascular Disease Focus Group recommends starting with low doses of pravastatin or atorvastatin , which are least likely to interact with Protease Inhibitors. Lovastatin, simvastatin, and fluvastatin are metabolized by the same CP450 liver enzyme system as PIs, and concurrent use can lead to high drug levels and intensified side effects; these drugs are contraindcated with PIs. Statins may cause muscle toxicity.

- **Fibrates** are used to treat high triglycerides or hypercholesterolemia accompanied by high triglycerides. This class includes fenofibrate and gemfibrozil. According to the AACTG focus group, these drugs are unlikely to interact with antiretroviral drugs, but this has not been well studied and they should be used with caution.

- **Bile sequesterants** such as cholestyramine and colestipol are used to decrease cholesterol levels; however, in some cases these drugs have been associated with increased triglyceride levels, and their interactions with antiretroviral drugs have not been well studied. The AACTG focus group discourages their use in people receiving HAART.

- **Nicotinic acid** (niacin) is also used to lower LDL cholesterol. Side effects include skin flushing and itching. The drug also can cause insulin resistance, and the AACTG focus group does not recommend it for people taking antiretroviral drugs that are themselves associated with insulin resistance.

Diabetes management

People taking HAART should have their blood sugar monitored regularly and be alert for early signs of diabetes such as frequent urination and increased hunger and thirst. To manage insulin resistance and diabetes, AACTG focus recommend that diet modification and increased exercise be tried first, followed by oral anti-diabetes agents such as the glitazone drugs and metformin . Diabetes drugs should be used with caution in people taking antiretroviral drugs.

Blood Pressure Control

Diet modification and exercise should be the first steps in controlling hypertension. Sodium consumption can lead to high blood pressure, and the NCEP recommends no more than 2,400 mg per day. If these measures are inadequate, hypertension-reducing drugs including ACE inhibitors, beta blockers, calcium channel blockers, diuretics, and vasodilators -- may be used. According to the AACTG focus group, no class of antihypertensives is completely contraindicated with HAART, but calcium channel blockers should be used with caution due to potential interactions with PIs.

Changes in Antiretroviral Therapy

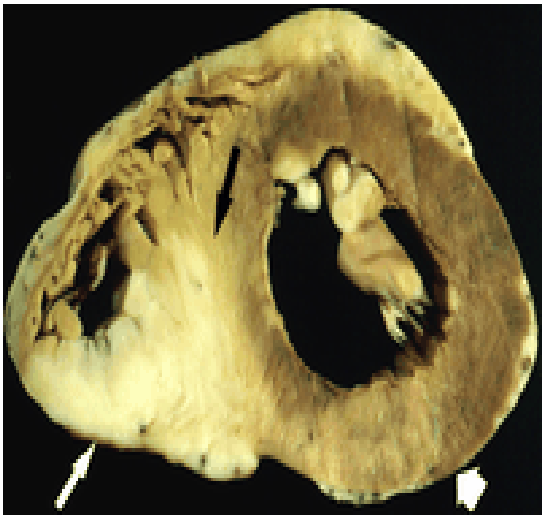
Switching to a protease-sparing regimen or to a PI that is less likely to cause metabolic abnormalities may be done in conjunction with or instead of lipid-lowering drugs. Drs. Falusi and Aberg recommed that providers initially attempt to manage hyperlipidemia without switching HAART regimens, especially if the person is adherent, is otherwise tolerating their drugs, and has good virologic control. In people with existing cardiovascular risk factors, however, it may be prudent to start with or switch to a regimen with fewer metabolic side effects.

CARDIAC TUMOURS

Various malignancies are associated with AIDS both in adults and children. Kaposi sarcoma, non-Hodgkin lymphoma, squamous cell carcinoma, Hodgkin disease, and leiomyosarcoma (in children) are the more prevalent malignancies and are found with increasing frequency in patients infected with HIV[79]. Of these malignancies, Kaposi Sarcoma and non-Hodgkin lymphoma most often affect the heart.

Kaposi Sarcoma, which is caused by human herpesvirus 8, is the most common cardiac tumor seen in patients with AIDS. It was probably the first cardiac complication of AIDS to be recognized: Autran et al reported a case of Kaposi sarcoma of the heart in a young Haitian woman with AIDS in 1983. Kaposi sarcoma is a low-grade malignancy that derives from mesenchymal or endothelial cells the most common AIDS-related neoplasm, it occurs in approximately 30% of patients, mostly in homosexual or bisexual males[79]. Cardiac involvement in Kaposi sarcoma in patients with AIDS usually occurs as part of disseminated disease. The epicardium and pericardium are the most frequently involved cardiac structures. Coronary wall infiltration and myocardial involvement also may occur. Pericardial hemorrhage with fatal cardiac tamponade has been described as a complication[80]. In 7% of cases of cardiac tamponade in HIV-infected patients, Kaposi sarcoma was the cause. Isolated Kaposi sarcoma of the heart is rare.

Lymphoma is the second most common tumor involving the heart in AIDS patients. Non-Hodgkin lymphoma was added to the group of AIDS-defining illnesses in 1987, after a 60- to 100-fold increase in the rate of lymphoma was observed among patients with HIV. Lymphoma is the first manifestation of AIDS in up to 4% of new cases. Primary cardiac malignancy associated with HIV infection is generally due to cardiac lymphoma[81]. In 80%–90% of cases, the majority of such lesions are high-grade B-cell tumors that consist of large cell immunoblastic or small non-cleaved-cell (Burkitt or Burkitt-like) lymphomas. At gross specimen examination, they may appear as a single mass or as multiple firm nodules of white tissue within the myocardium[81]. Any cardiac chamber may be affected, but the right atrium is most commonly involved, followed by the right ventricle, left ventricle, left atrium and atrial and ventricular septa. Pericardial extension is common. Secondary cardiac involvement in extra nodal lymphoma may manifest as a mediastinal mass with extension through the pericardium or as lymphangitic or hematogenous lesions. Chest radiographs of patients with cardiac lymphoma usually show cardiomegaly, pericardial effusion, and signs of congestive heart failure. Echocardiograms typically depict a hypoechoic cardiac mass with or without pericardial effusion. On unenhanced CT images, the tumors have soft-tissue attenuation equal to or less than that of myocardium.



Non-Hodgkin lymphoma in a young man with AIDS. Gross autopsy specimen shows extensive infiltration in the right ventricle (long white arrow), interventricular septum (black arrow), and inferior wall of the left ventricle (short white arrow).

Contrast-enhanced CT scan in a 28 years old man with AIDS shows abnormal irregular thickening of the lateral wall of the left ventricle (arrow) and interatrial wall (arrowheads).

THINGS TO KNOW

1. Use of antiretroviral therapy (ART) has decreased overall mortality associated with HIV remarkably, but cardiovascular disease (CVD) accounts for a growing proportion of the deaths among HIV patients.

2. The relative risk of MI in HIV compared to non-HIV subjects is increased approximately 1.7-1.8 fold. This excess risk may increase further with advancing age.

3. HIV disease itself is associated with dyslipidemia (primarily increases in triglycerides and reductions in HDL).

4. Studies have shown the increase in CVD among HIV-infected patients is associated with the use of specific ARTs, metabolic abnormalities related to ART use such as diabetes and dyslipidemia and, importantly, traditional risk factors including smoking which is prevalent among HIV infected individuals.

5. There is significant variation with respect to cardio vascular effects in a given antiretroviral class. The contributions of these changes to cardiovascular risk may vary depending on the

presence of other risk factors. Various ART treatments have been shown to increase triglyceride levels, reduce glucose disposal, contribute to insulin resistance, result in mitochondrial dysfunction, lead to subcutaneous fat loss and lead to insulin resistance.

6. Despite the association of ART use with CVD, the long-term effects of continuous ART are unknown. Continuous suppression of viral load may have additional benefits beyond the known clinical advantages, such as reduction of inflammation or other undefined beneficial effects. Further research is needed to answer this important question.

7. The Framingham Risk Score is reasonably accurate in predicting CVD events in patients with HIV but may under-predict MI's among the large group of HIV infected patients who smoke. Prediction might be improved upon using HIV specific equations, but these have not yet been validated.

8. Progressive LV dilation in HIV infected children without adequate compensatory hypertrophy results in excessive LV

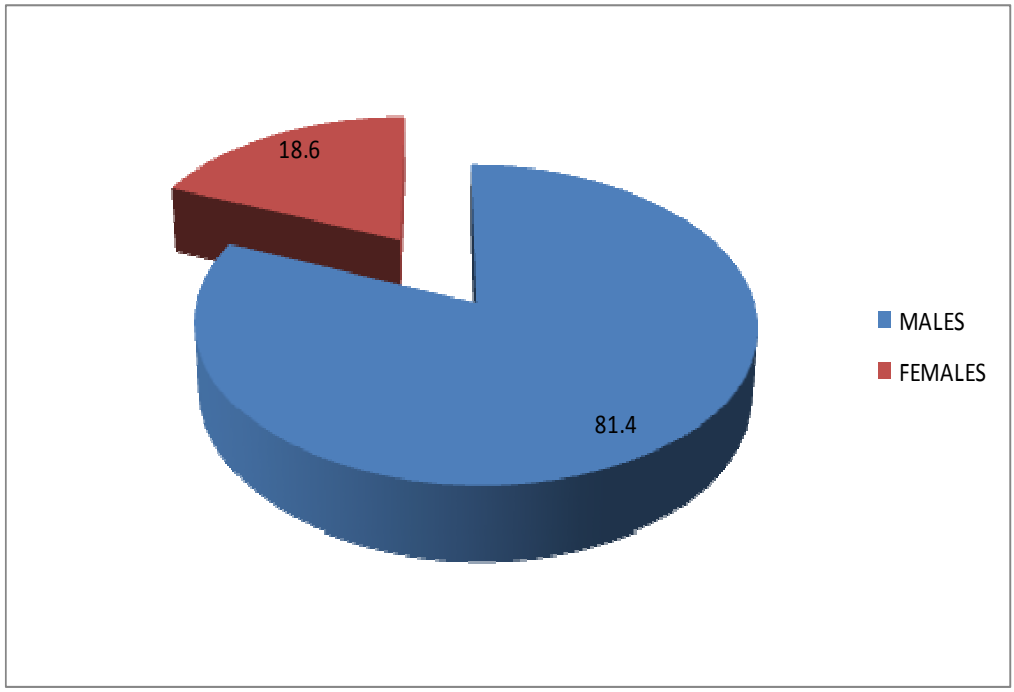
afterload, reduced LV function, and symptomatic cardiovascular disease.

9. Nonatherosclerotic heart disease, including pulmonary hypertension, pericardial disease and reduced cardiac contractility may result from both HIV infection and ART therapy.

10. Strategies to prevent CVD in HIV infected patients should focus on reduction of traditional risk factors, as well as HIV and ART specific risk factors. Because of virological resistance that may develop due to changes in ART, caution should be exercised in changing individual drugs and the balance of risk to benefit must be assessed. Use of agents for lipid lowering, insulin sensitization, and reduction of central adiposity as well as lifestyle strategies such as smoking cessation may improve individual CV risks in the HIV population[81].

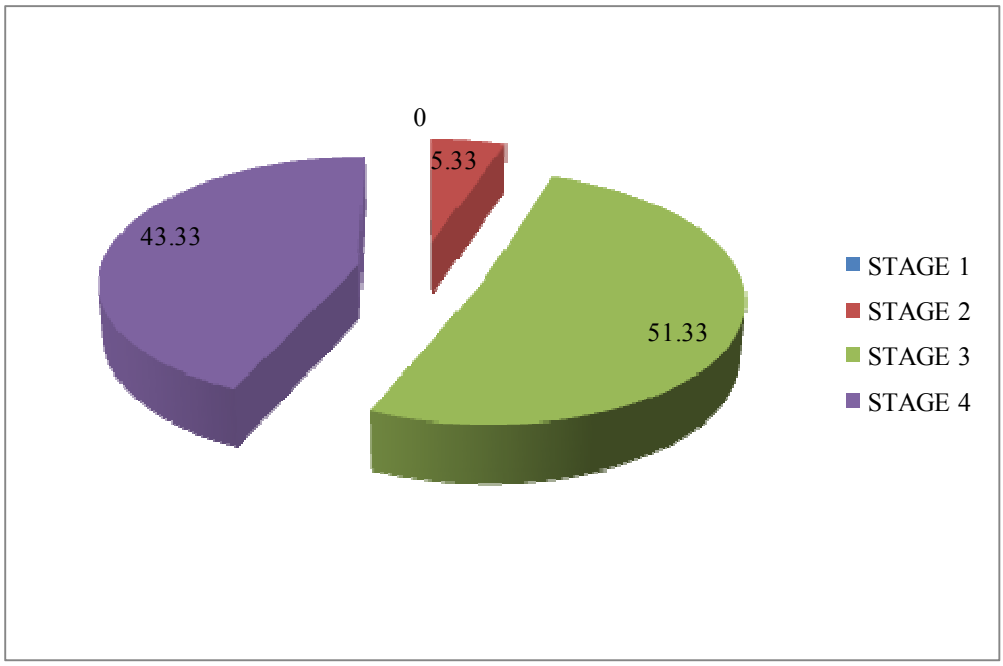
RESULTS AND OBSERVATION

PERCENTAGE OF MALES AND FEMALES



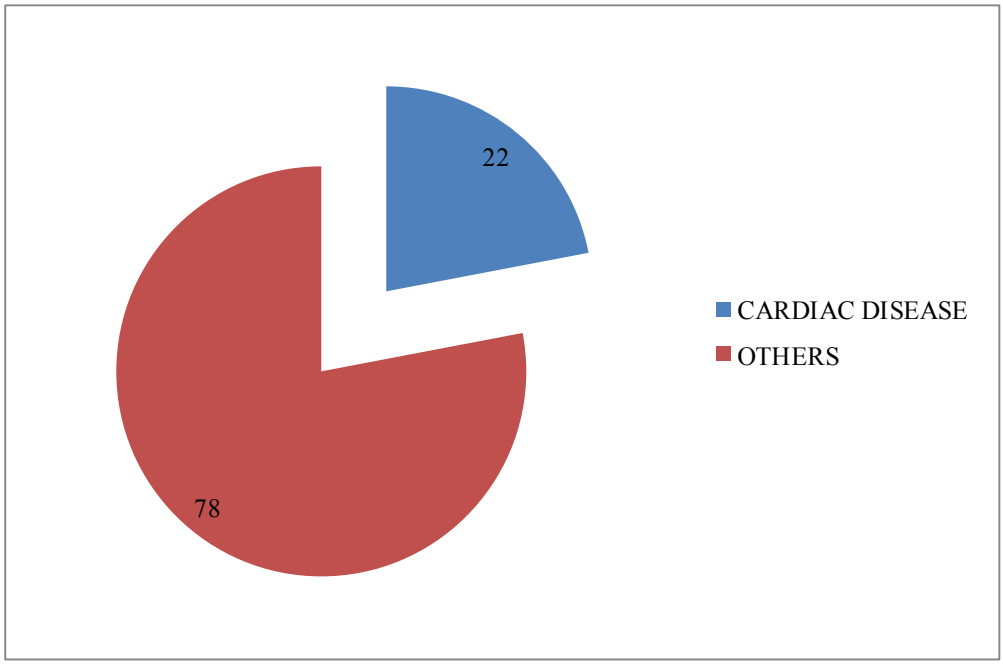
	No of Patients	Percentage
MALES	137	81.4%
FEMALES	13	18.6%

PERCENTAGE OF VARIOUS CLINICAL STAGES OF HIV ANALYSED



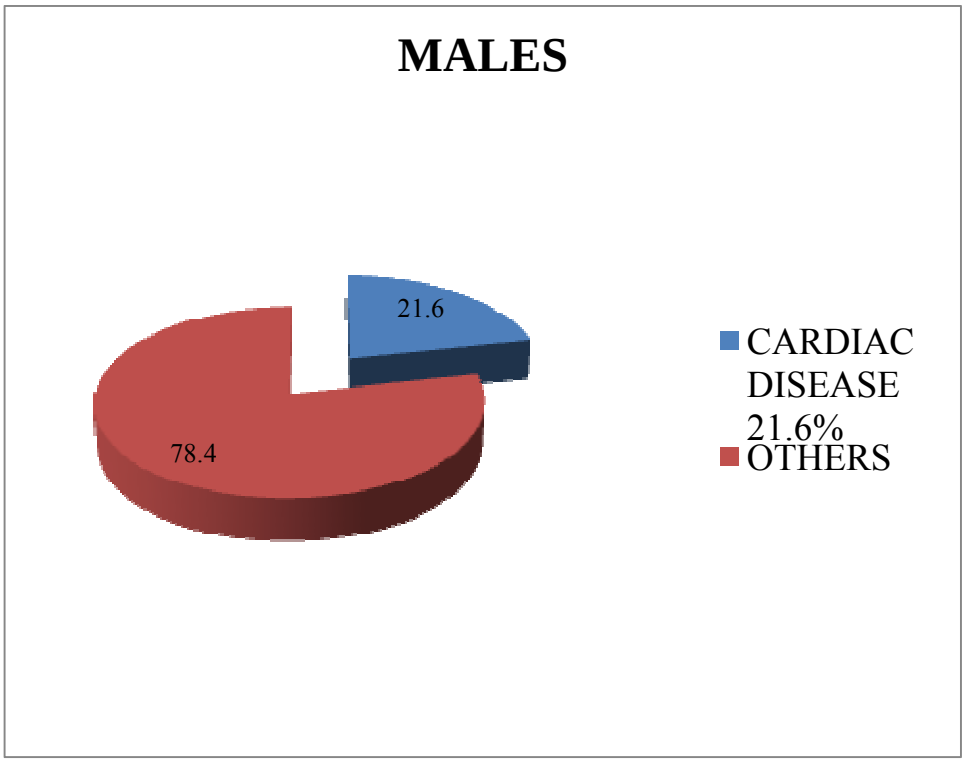
	No of Patients	Percentage
STAGE 1	0	0%
STAGE 2	8	5.33%
STAGE 3	77	51.33%
STAGE 4	65	43.33%

PERCENTAGE OF CARDIAC DISEASE



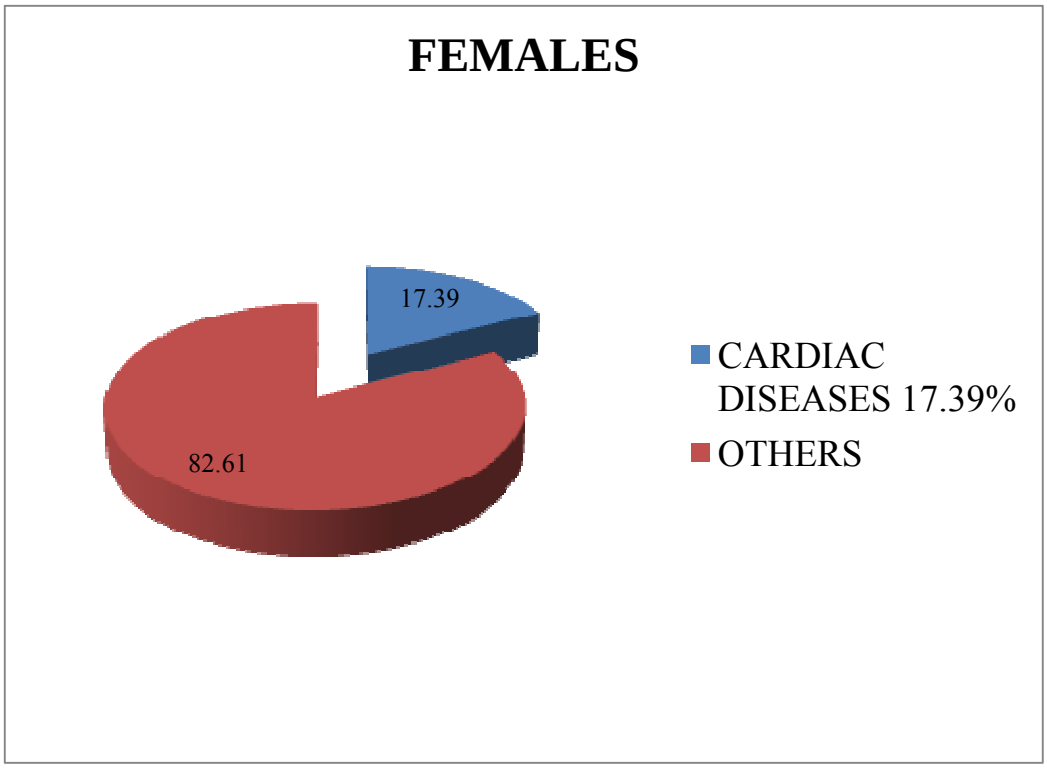
	No of Patients	Percentage
Cardiac Disease	33	22%
Others	117	78%

CARDIAC DISEASES IN MALES:



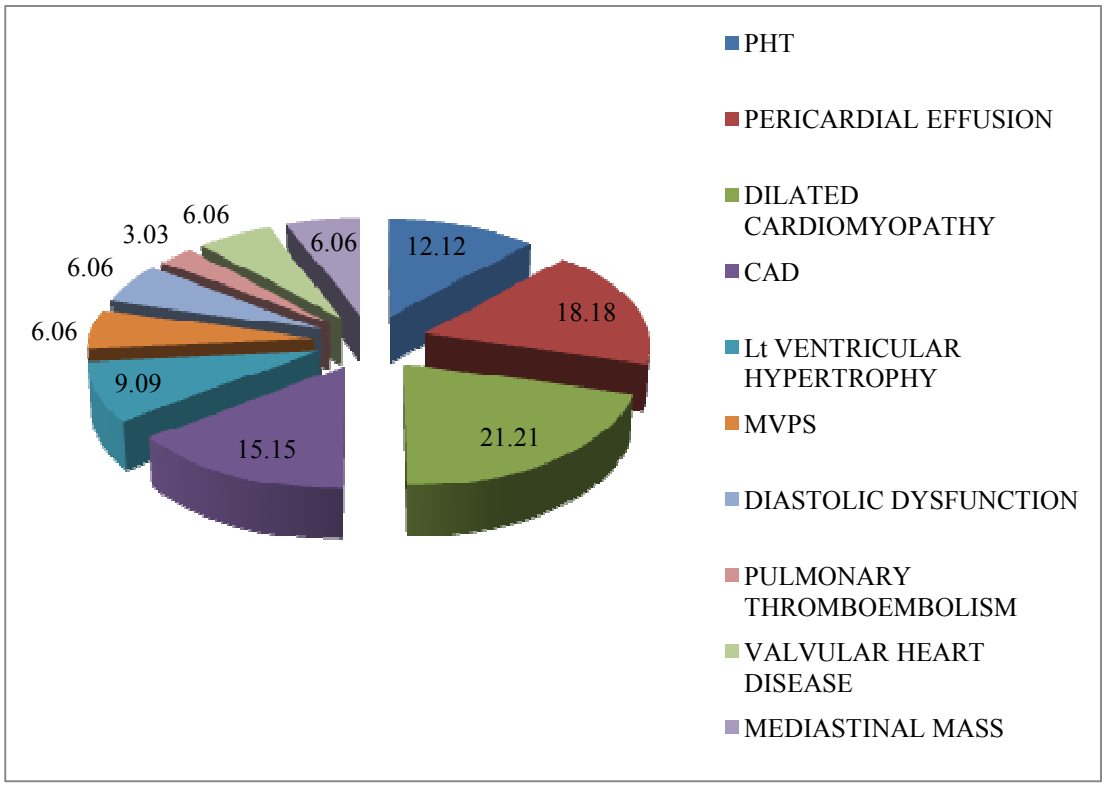
	No of Patients	Percentage
Cardiac Disease	30	21.6%
Others	107	78.4%

CARDIAC DISEASES IN FEMALES



	No of Patients	Percentage
Cardiac Disease	4	17.39%
Others	9	82.61%

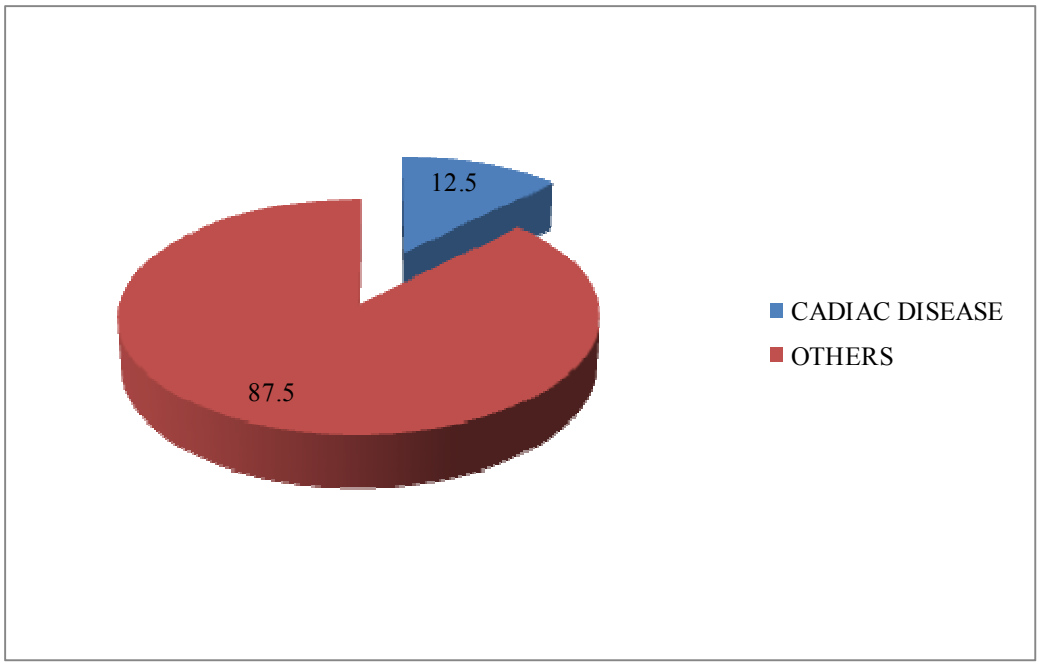
PERCENTAGE OF VARIOUS CARDIAC DISEASES IN HIV



Cardiac Manifestations	No. of Patients	%
Pericardial Effusion	6	18.18%
Cardiomyopathy	7	21.21%
Pulmonary Hypertension	4	12.12%
Left Ventricular Hypertrophy	3	09.09%
CAD	5	15.15%
Diastolic Dysfunction	2	06.06%
MVPS	2	06.06%
Valvular Heart Disease	2	06.06%
Pulmonary Thrombo Embolism	1	03.03%
Mediastinal Mass	2	06.06%

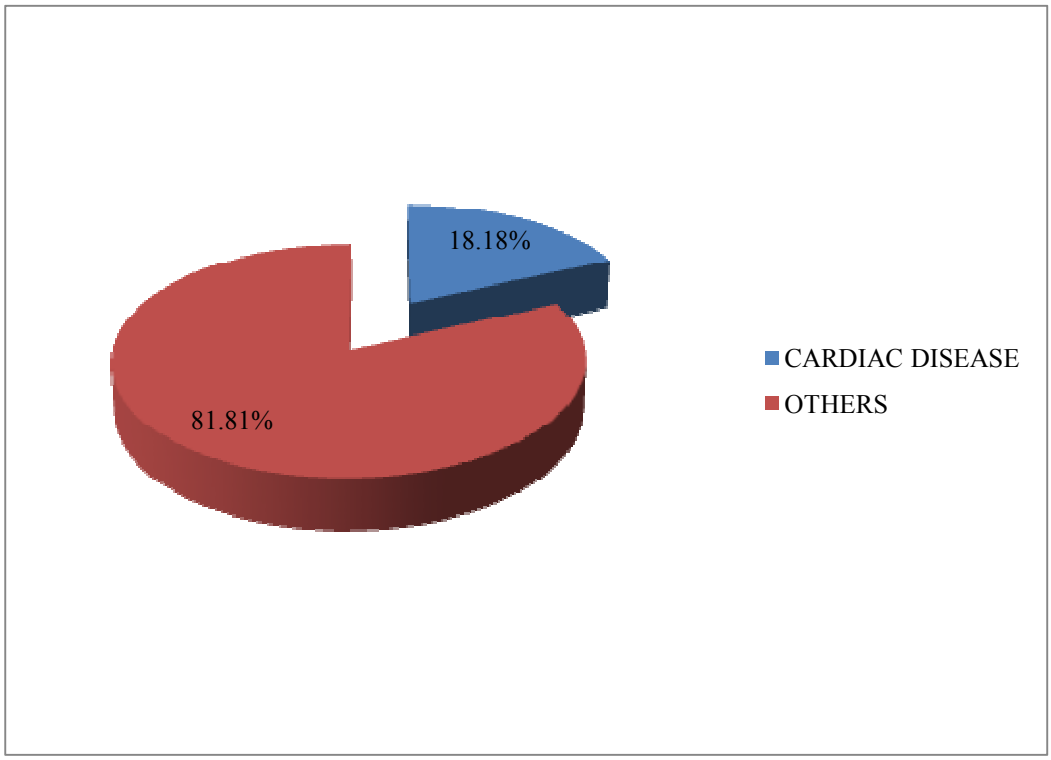
CARDIAC MANIFESTATIONS IN VARIOUS STAGES OF HIV

STAGE 2



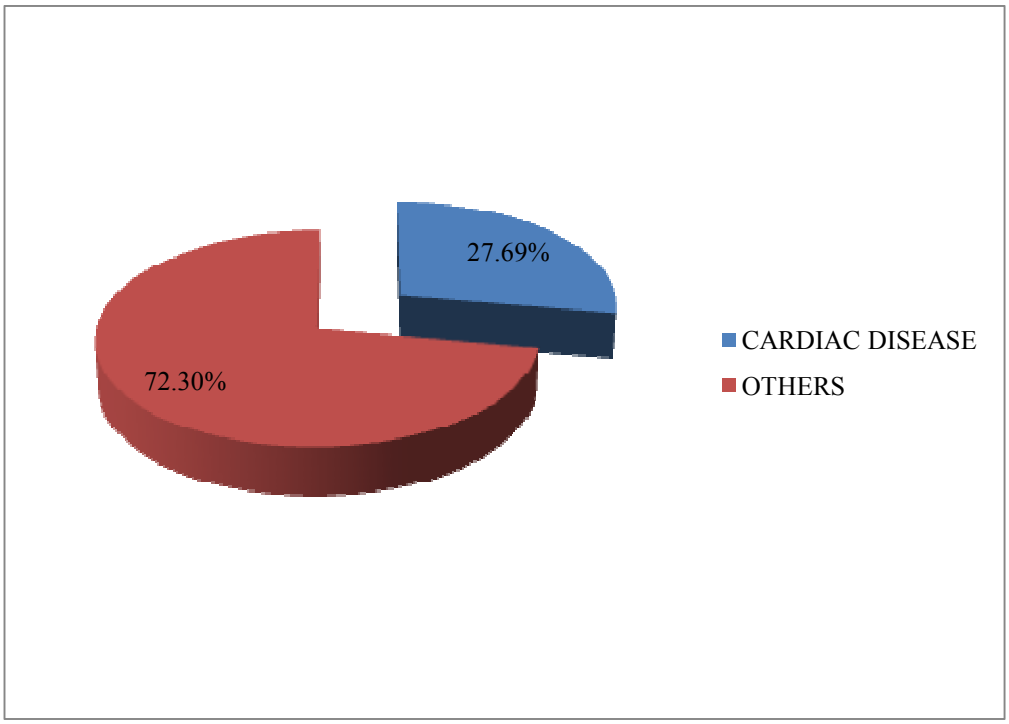
	No of Patients	Percentage
Cardiac Disease	1	12.5%
Others	7	87.5%

STAGE 3



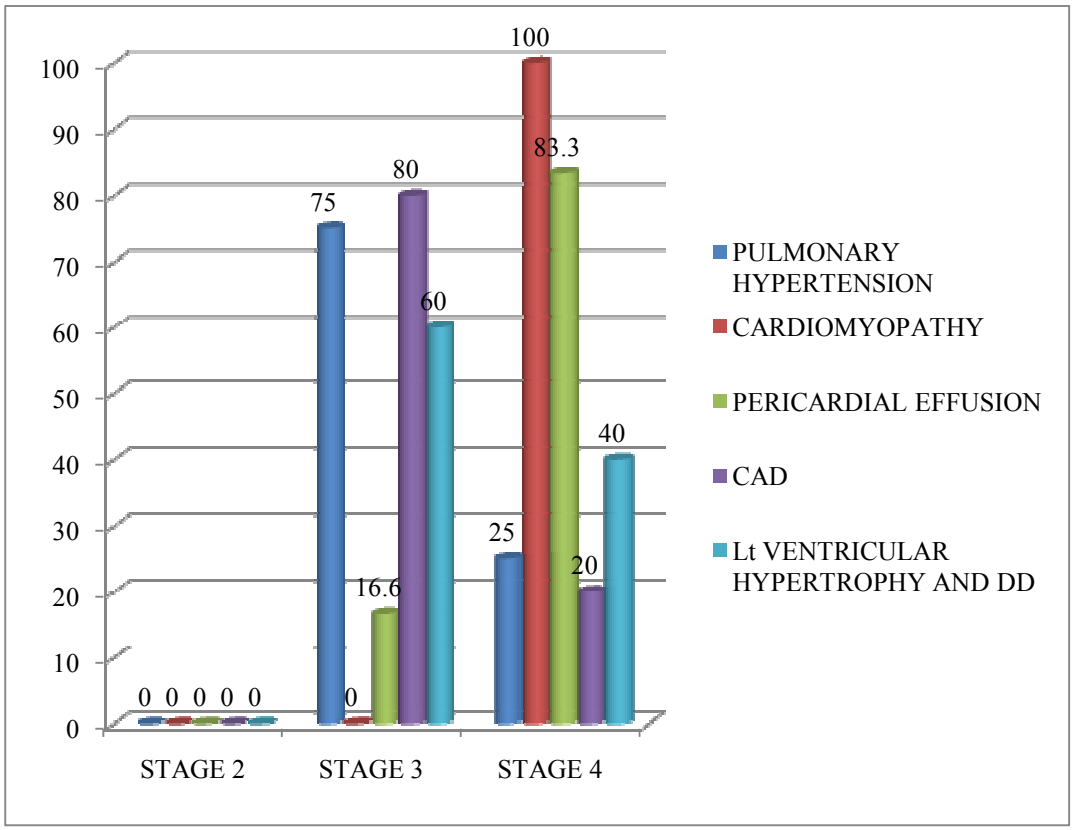
	No of Patients	Percentage
Cardiac Disease	14	18.18%
Others	63	81.81%

STAGE 4



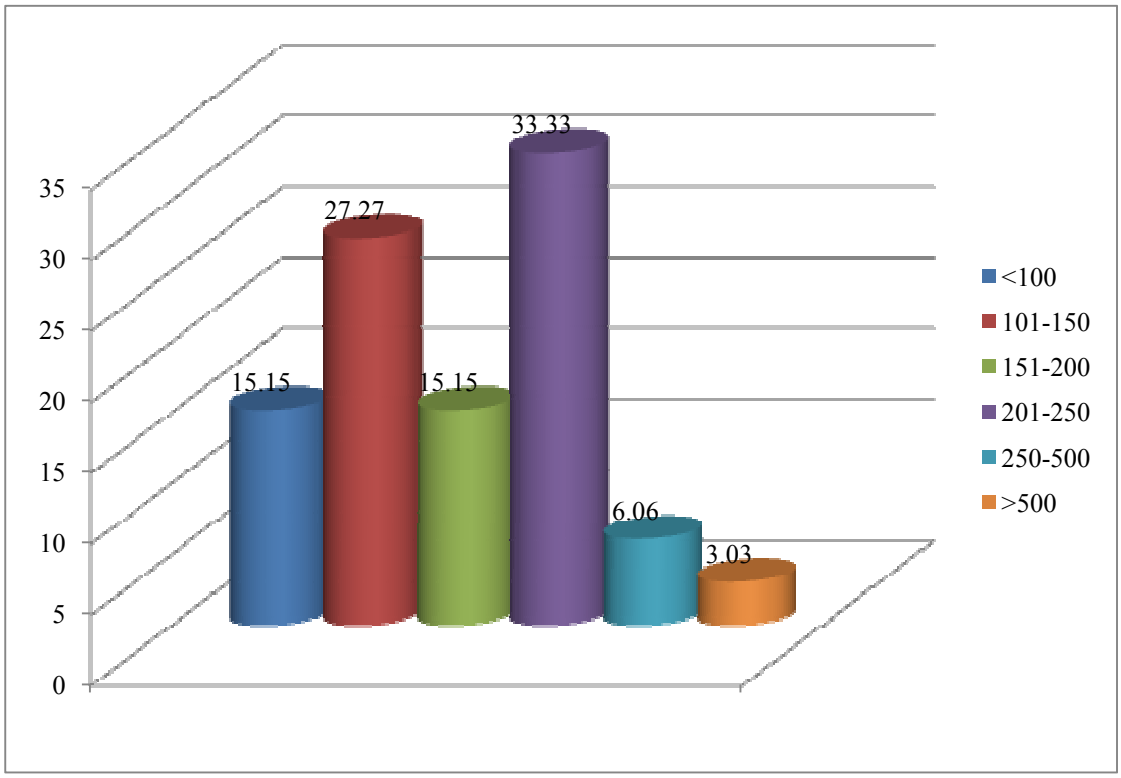
	No of Patients	Percentage
Cardiac Disease	18	27.69%
Others	47	72.30%

COMPARISION BETWEEN VARIOUS STAGES



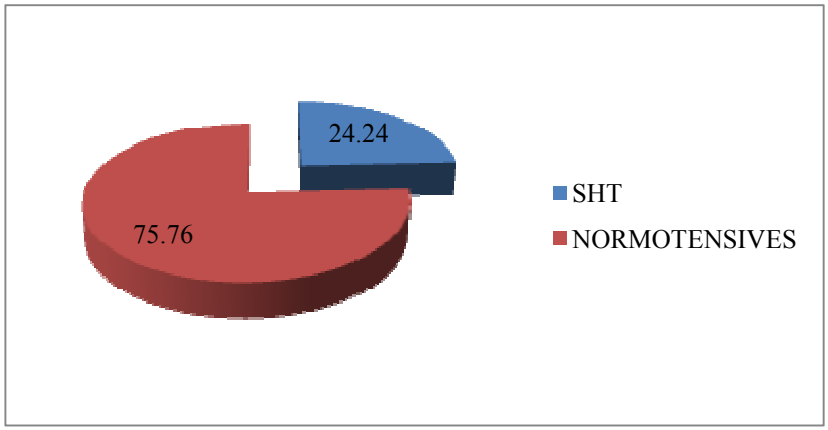
CARDIAC DISEASE	STAGE 3		STAGE 4	
PHT	3	75%	1	25%
Cardiomyopathy	0	0%	7	100%
Pericardial Effusion	1	16.6%	5	83.3%
CAD	4	80%	1	20%
LVH and DD	3	60%	2	40%

INCIDENCE OF CARDIAC DISEASES TO CD_4 COUNT

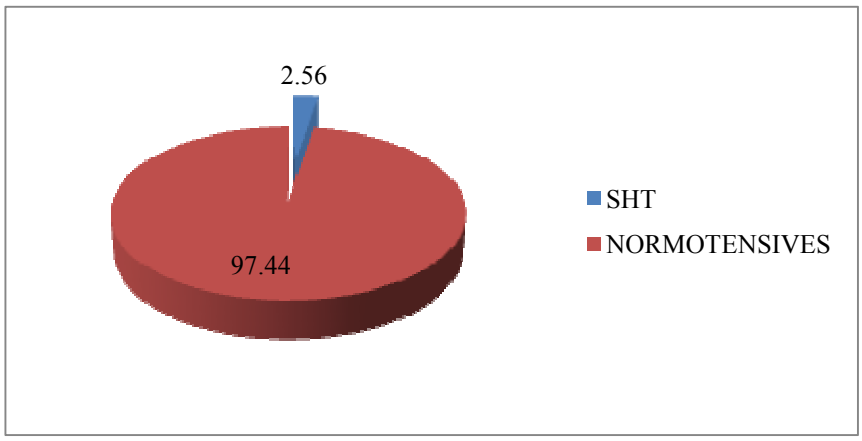


CD_4 COUNT	NO. OF PATIENTS	PERCENTAGE
<100	5	15.15%
101-150	9	27.27%
151-200	5	15.15%
201-250	11	33.33%
251-500	2	06.06%
>500	1	03.03%

PREVALENCE OF SYSTEMIC HYPERTENSION WITH CARDIAC DISEASES IN HIV

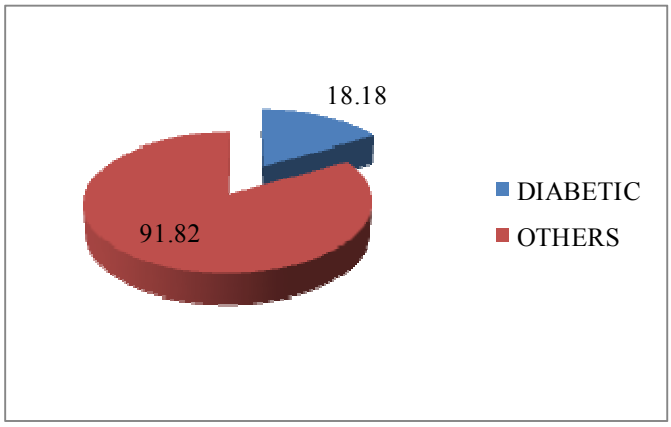


PREVALENCE OF SHT WITHOUT CARDIAC DISEASE IN HIV

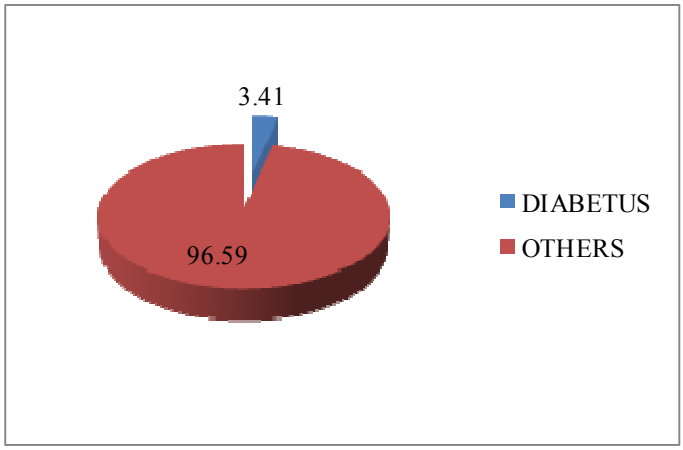


	No of Patients	Percentage
With Cardiac Disease	8	24.24%
Without Cardiac Disease	3	2.56%

PREVALENCE OF DIABETES WITH CARDIAC DISEASES IN HIV



PREVALENCE OF DIABETES WITHOUT CARDIAC DISEASE IN HIV

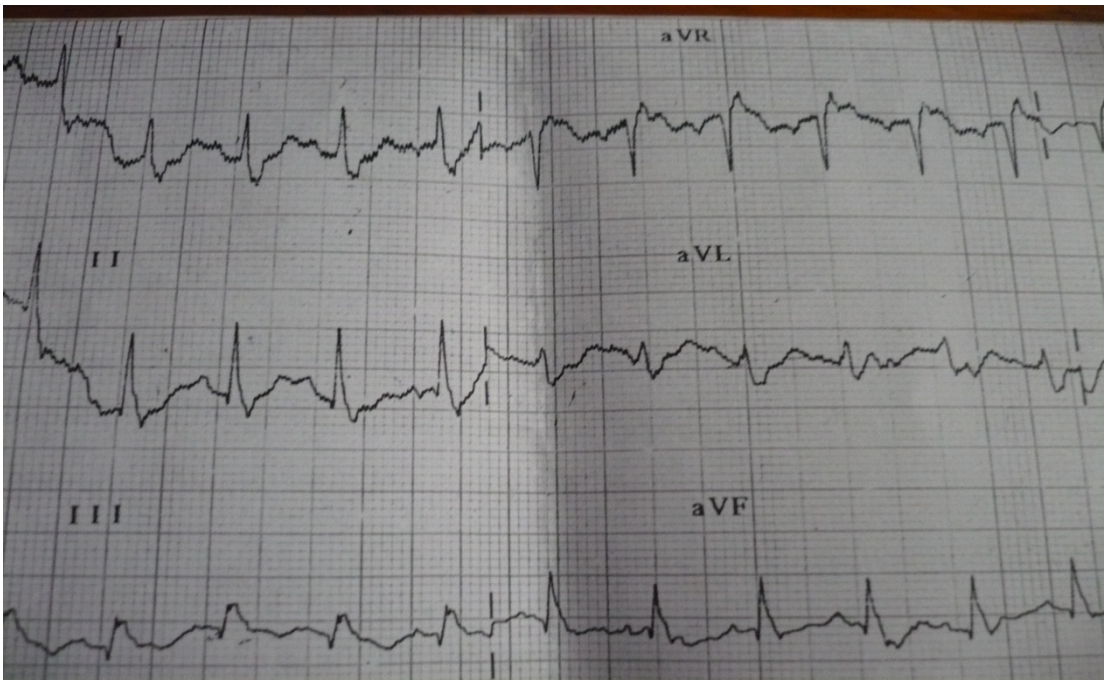


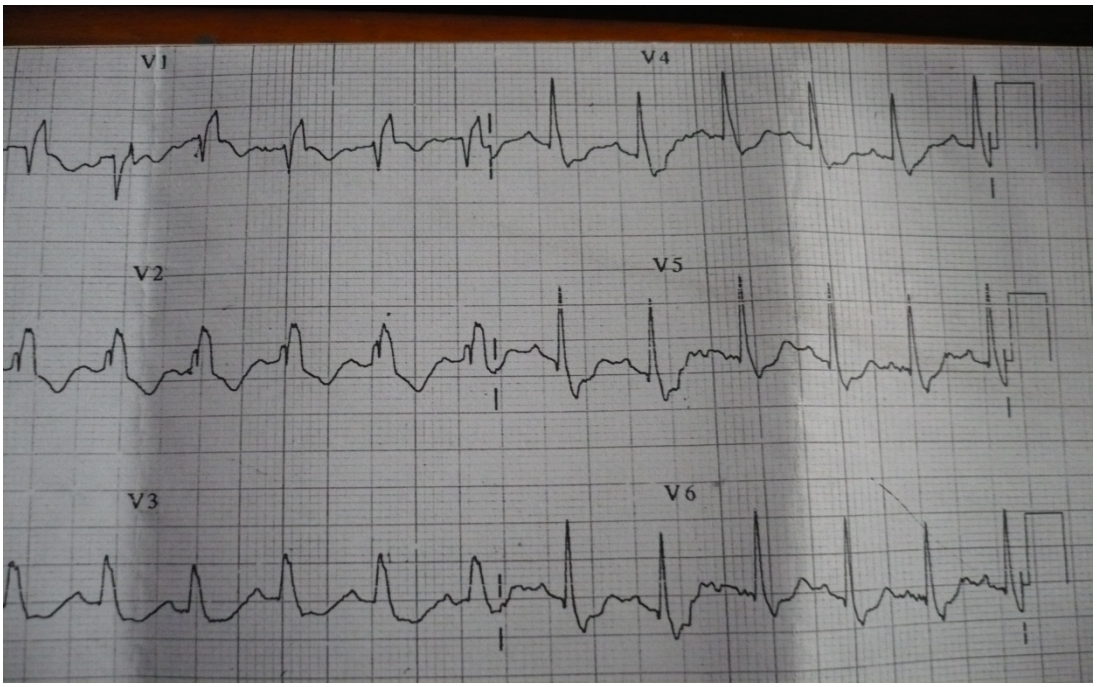
	No of Patients	**Percentage**
With Cardiac Disease	6	18.18%
Without Cardiac Disease	4	3.41%

INTERESTING ECGS ENCOUNTERED

ST, rate 120, low voltage, electrical alternans, findings diagnostic of pericardial effusion

ST S1Q3T3 PATTERN RBBB T WAVE INVERSION IN V_1-V_3 SUGGESTIVE OF PULMONARY THROMBOEMBOLISM





DISCUSSION

In this study echocardiographic manifestations of 150 HIV patients were analysed irrespective of their CD_4 count, stage of disease and ART status. Among them 91.4% are males and 18.6% are females.

They were staged according to WHO clinical staging of HIV/AIDS. 5.33% belong to stage 2, 51.33% belong to stage 3 and 43.33% belong to stage 4.

The overall incidence of cardiac manifestation is 22% among the HIV patients.

The incidence of cardiac manifestation in males is found to be 21.6% whereas it is 17.39% in females. Thus the incidence is found to be higher in males when compared to females.

Among the cardiac manifestations the most common is dilated cardiomyopathy 21.21% followed by pericardial effusion 18.18%. Incidence of other manifestations is coronary artery disease 15.15%, pulmonary hypertension 12.12%, left ventricular hypertrophy 9.09%, diastolic dysfunction 6.06%, mitral valve prolapse 6.06%, other valvular

heart disease 6.06%, pulmonary thromboembolism 3.03% and mediastinal mass 6.06%.

Comparing various stages of HIV, in stage 2 one patient is found to have mitral valve prolapse syndrome. In stage 3 the incidence is 18.18% and the incidence in stage 4 is 27.69%. This shows that incidence of cardiovascular manifestation increases as the disease progresses.

When individual manifestations are compared, incidence of dilated cardiomyopathy and pericardial effusion is found to be higher in stage 4. The incidence is 100% and 83.3% respectively. Whereas the incidence of pulmonary hypertension, coronary artery disease, left ventricular hypertrophy and diastolic dysfunction is found to be more in stage 3. The incidences are 75%, 80% and 60% respectively.

Almost 90.9% of manifestations occur when CD_4 count is below 250.

On analyzing the risk factors for cardiac disease 67.15% of males consume alcohol and 64.23% of males smoke. All the females deny smoking and alcohol history.

Incidence of systemic hypertension is 24.24% in patients with cardiac disease and in patients without cardiac disease is 2.56% .

Incidence of diabetes mellitus is 18.18% in patients with cardiac disease and in patients with cardiac disease is 3.41%. This shows that the presence of other risk factors like systemic hypertension and diabetes increases the incidence of cardiac diseases.

CONCLUSION

- Cardiac involvement and cardiovascular complications are commonly seen in HIV-infected patients.
- As the epidemic progresses and new treatments help increase the long-term survival of affected individuals, cardiovascular complications will become more common.
- The common cardiovascular manifestation seen in HIV patients is dilated cardiomyopathy.
- The incidence of cardiovascular manifestations increases as the disease progresses.
- The presence of risk factors like systemic hypertension and diabetes further increases the risk of cardiac diseases.
- Strategies to prevent cardiovascular disease in HIV-infected patients should focus on reducing traditional risk factors, as well as HIV and ART-specific risk factors.
- Early recognition and prompt treatment are important to prevent significant morbidity from cardiac involvement. Whether this

approach will prolong survival in AIDS patients remains to be seen.

- It's important to stratify CVD risk in HIV-infected patients. Clinicians can use existing general risk stratification algorithms, such as the Framingham Risk Score, to measure HIV patients risk for heart disease.

- In essence, it means practicing good preventive medicine with all patients, including those who have HIV.

BIBLIOGRAPHY

1. *AmatiG*, di Gioia CR, Gallo P. Pathological findings of HIV-associated cardiovascular disease. *Ann N Y Acad Sci 2001*;*946*:*23*–45.

2. Enger C, Graham N, Peng Y, et al. Survival from early, intermediate, and late stages of HIV infection. *JAMA.* 1996;275:1329-1334.

3. Monsuez JJ, Vittecoq D, Kinney EL. Increased survival of AIDS patients with heart disease within 10 years? *Circulation.* 1996;94: 2312-2313.

4. Mocroft A, Youle M, Morcinek J, et al. Survival after diagnosis of AIDS: a prospective observational study of 2625 patients. *BMJ.* 1997;314:409-413.

5. Autran B, GorinI, Leibowitch M, et al. AIDS in a Haitian woman with cardiac Kaposi's sarcoma and Whipple's disease. *Lancet.* 1983;1: 767-768.

6. Grody WW, Cheng L, Lewis W. Infection of the heart by the human immunodeficiency virus. *Am J Cardiol* 1990;66:203-6.

7. Lipshultz SE, Fox CM, Perez-Atayde AR, et al. Identification of human immunodeficiency virus-1 RNA and DNA in the heart of a child with cardiovascular abnormalities and congenital acquired immunodeficiency syndrome. *Am J Cardiol* 1990;66:246-51.

8. Codriquez ER, Nasim S, Hsia J, et al. Cardiac myocytes and dendritic cells harbor human immunodeficiency virus in infected patients with and without cardiac dysfunction: detection by multiplex, nested, polymerase chain reaction in individually microdissected cells from right ventricular endomyocardial biopsy tissue. *Am J Cardiol* 1991;68:1511-20

9. Francis CK. Cardiac involvement in AIDS. *CurrProblCardiol* 1990;15:574-639.

10. Eisenberg MJ, Gordon AS, Schiller NB. HIV associated pericardial effusion. *Chest* 1992;102:956-8.

11. Turco M, Seneff M, McGrath BJ, Hsia J. Cardiac tamponade in the acquired immunodeficiency syndrome. *Am Heart J* 1990;120:1467-8.

12. Scarpella EG, Fatmi AA, Brito MA. Bacterial pericarditis and cardiac tamponade in patients infected with HIV-1: case report and review. *Clin Infect Dis* 1995;21:1518-9.

13 Nathan PE, Arsura EL, Zappi M. Pericarditis with tamponade due to cytomegalovirus in the acquired immunodeficiency syndrome.*Chest* 1991;99:765-6.

14. Freedberg RS, Gindea AJ, Dietrich DT, et al. Herpes simplex pericarditis in AIDS.*N Y State J Med* 1987;87:304-6.

15. Toma E, Poisson M, Claessens MR, et al. Herpes simplex type 2 pericarditis and bilateral facial palsy in patients with AIDS [letter]. *J Infect Dis* 1989;160:553-4.

16. Bernard SA. Cardiac manifestations. In Libman H, Witzburg RA, eds. *HIV infection: a primary care manual.* Boston: Little, Brown, 1996:123-37.

17. Stechel RP, Cooper DJ, Greenspan J, et al. Staphylococcal pericarditis in a homosexual patient with AIDS-related complex. *N Y State J Med* 1986;86:592-3.

18. Decker CF, Tuazon CU. Staphylococcus aureus pericarditis in HIV-infected patients. *Chest* 1994;101:1461-3.

19. Scarpella EG, Fatmi AA, Brito MA. Bacterial pericarditis and cardiac tamponade in patients infected with HIV-1: case report and review. *Clin Infect Dis* 1995;21:1518-9.

20. Dalli E, Quesada A, Juan A, et al. Tuberculosis pericarditis as the first manifestations of acquired immunodeficiency syndrome. *Am Heart J* 1987;114:905-6.

21. D'Cruz IA, Sengupta EE, Abraham C, et al. Cardiac involvement including tuberculosis pericardial effusion, complicating acquired immunodeficiency syndrome. *Am Heart J* 1986;112:1100-2.

22. Schuster M, Valentine F, Holtzman R. *Cryptococcus* pericarditis in an IVDA. *J Infect Dis* 1985;152:842.

23. Brivet P, Livartowski J, Herve P, et al. Pericardial cryptococcal infection in acquired immunodeficiency syndrome. *Am J Med* 1987;82:1273.

24. Ziegler JL, Bragg K, Abrams D, et al. High-grade non-Hodgkin's lymphoma in patients with AIDS. *Ann N Y AcadSci* 1984;437:412-9.

25. Ioachim HL, Cooper MC, Hellman GC. Lymphomas in men at high-risk for acquired immunodeficiency syndrome (AIDS): a study of 21 cases. *Cancer* 1985;56:2831-42.

26. Kaul S, Fishbein MC, Siegel RJ. Cardiac manifestations of acquired immunodeficiency syndrome: a 1991 update. *Am Heart J* 1991;122:535-44.

27. Bernard SA. Cardiac manifestations. In Libman H, Witzburg RA, eds. *HIV infection: a primary care manual.* Boston: Little, Brown, 1996:123-37.

28. Cheitlin MD. Cardiac involvement in HIV disease. In Cohen PT, Sande MA, Volberding PA, eds. *The AIDS knowledge base.* Boston: Little, Brown, 1994.

29. DeCastro S, Migliau G, D'Amati G, et al. Heart involvement in AIDS: a prospective study during various stages of the disease. *Eur Heart J* 1992;13:1452-9.

30. Hsai J, Ross AM. Pericardial effusion and pericardiocentesis in human immunodeficiency virus infection.*Am J Cardiol* 1994;74:94-6

31. Currie PF, Boon NA. Cardiac involvement in human immunodeficiency virus infection [editorial].*Q J Med* 1993;86:751-3.

32. Jacob AJ, Sutherland G, Bird AG, et al. Myocardial dysfunction in patients infected with HIV: prevalence and risk factors. *Br Heart J* 1992;68:549-53

33. Acierno LJ. Cardiac complications in acquired immunodeficiency syndrome (AIDS): a review. *J Am CollCardiol* 1989;13:1144-54.

34. Patel RC, Frishman WH. Cardiac involvement in HIV infection. *Med Clin North Am* 1996;80:1493-1512.

35. Rose NR, Herskowitz A, Neumann DA. Autoimmunity in myocarditis models and mechanisms.*ClinImmunolImmunopathol* 1993;68:95-9.

36. Newman DA, Lane JR, Allen GS, et al. Viral myocarditis leading to cardiomyopathy: do cytokines contribute to pathogenesis? *ClinImmunolImmunopathol* 1993;68:181-90.

37. Lieberman EB, Herskowitz A, Rose NR, et al. A clinicopathological description of myocarditis. *ClinImmunolImmunopathol* 1993;68:191-6.

38. Hastillo A, Willis HE, Hess ML. The heart as a target organ of immune injury.*CurrProblCardiol* 1991;16:377-442.

39. Kennedy JR. Does HIV disrupt a naturally occurring immune modulation system? *Med Hypotheses* 1993;41:445-9.

40. Herskowitz A, Willoughby S, Wu TC. Immunopathogenesis of HIV-1 associated cardiomyopathy. *ClinImmunolImmunopathol* 1993;68:234-41.

41. Herskowitz A, Neumann DA, Ansari AA. Concepts of autoimmunity applied to idiopathic dilated cardiomyopathy. *J Am CollCardiol* 1993;22:1385-8.

42. Kavanaugh-McHugh, Rowe S, Benjamin Y, Hutton N, Modlin J, Ruff A. Selenium deficiency and cardiomyopathy in malnourished pediatric AIDS patients [abstract]. V International Conference on AIDS, Montreal, 1989:Section B: 329.

43. Zazzo JF, Chalas J, LaFont A, et al. Is nonobstructivecardiopathy in AIDS a selenium deficiency-related diseases? *JPEN J Parenter Enteral Nutr* 1988;12:537-8.

44. Dworkin BM. Selenium deficiency in HIV infection and the acquired immunodeficiency syndrome (AIDS). *Chem-Biol Interact* 1994;91:181-6.

45 Wynne J, Braunwald E.The cardiomyopathies and myocarditis. In Braunwald E, ed. *Heart disease: a text book of cardiovascular medicine.* Philadelphia: WB Saunders, 1997;41:1401-64.

46. Calabrese LH, Proffitt MR, Yen-Lieberman B, Hobbs RE, Ratliff NB. Congestive cardiomyopathy and illness related to the acquired immunodeficiency syndrome (AIDS) associated with isolation of retrovirus from myocardium. *Ann Intern Med.* 1987;107:691-62.

47. Cotton P. AIDS giving rising to cardiac problems [letter]. *JAMA.* 1990;263:2149.

48. Grody WW, Cheng L, Lewis W. Infection of the heart by the human immunodeficiency virus. *Am J Cardiol.* 1990;66:203-206

49. Johnson HM, Torres BA, Soos JM. Superantigens: structure and relevance to human disease. *ProcSocExpBiol Med.* 1996;212: 99-109.

50. Anderson DW, Virmani R, Reilly JM, et al. Prevalent myocarditis at necropsy in acquired immunodeficiency syndrome. *J Am CollCardiol.* 1988;11:792-799.

51. Acierno LJ. Cardiac complications in acquired immunodeficiency syndrome (AIDS): a review. *J Am CollCardiol.* 1989;13 1144-1154.

52. Mesa RA, Edell ES, Dunn WF, Edwards WD. Human immunodeficiency virus infection and pulmonary hypertension. *Mayo Clin Proc.* 1998;73:37-44.

53. *MehtaNJ, Khan IA, Mehta RN, Sepkowitz DA. HIV-related pulmonary hypertension: analytic review of 131 cases. Chest 2000;118: 1133–1141.*

54. *BugnoneAN, Viamonte M Jr, Garcia H. Imaging findings in human immunodeficiency virus-related pulmonary hypertension: report of five cases and review of the literature. Radiology 2002;223:820–827.*

55. Currie PF, Sutherland GR, Jacob AJ, Bell JE, Brettle RP, Boon NA. A review of endocarditis in acquired immunodeficiency syndrome and human immunodeficiency virus infection. *Eur Heart J.* 1995;16(suppl B):15-18

56. Cammarosano C, Lewis W. Cardiac lesion in acquired immune deficiency syndrome (AIDS). *J Am CollCardiol.* 1985;5:703-706.

57. Lopez JA, Ross RS, Fishbein MC, Seigel RJ. Nonbacterial thrombotic endocarditis.*Am Heart J.* 1987;113:773-784.

58. Rosen P, Armstrong O. Nonbacterial thrombotic endocarditis in patients with malignant neoplastic disease. *Am J Med.* 1973;54:23-29.

59. Patel RC, Frishman WH. Cardiac involvement in HIV infection. *Med Clin North Am.* 1996;80:1493-1512.

60. Nahass RG, Weinstein MP, Bartels J, Gocke DJ. Infective endocarditis in intravenous drug users: a comparison of human immunodeficiency virus type 1–negative and –positive patients. J Infect Dis. 1990;162:967-970.

61. Longo-MbenzaB, Tonduangu K, Seghers KV, Mubagwa D. HIV infection and pericardial disease invasion in Africa [in French]. Arch Mal Coeur Vaiss1997;90:1377–1384

62. MehtaNJ, Khan IA. HIV-associated coronary artery disease.Angiology2003;54:269–275.

63. Chetty R, Batitang S, Nair R. Large artery vasculopathy in HIV-positive patients: another vasculitic enigma. Hum Pathol2000;31:374–379.

64. MarksC, Kuskov S. Pattern of arterial aneurysms in acquired immunodeficiency disease. World J Surg1995;19:127–132.

65. MirzaH, Patel P, Suresh K, Krukenkamp I, Lawson WE. HIV disease and an atherosclerotic ascending aortic aneurysm.Rev Cardiovasc Med2004;5:176–181.

66. Hadigan C, Meigs JB, Rabe J, et al. Increased PAI-1 and tPA antigen levels are reduced with metformin therapy in HIV-infected patients with fat redistribution and insulin resistance. J ClinEndocrinolMetab2001;86:939–943.

67. WitzM, Lehmann J, Korzets Z. Acute brachial artery thrombosis as the initial manifestation of human immunodeficiency virus infection. Am J Hematol2000;64:137–139.

68. JacobsonMC, Dezube BJ, Aboulafia DM. Thrombotic complications in patients infected with HIV in the era of highly active antiretroviral therapy: a case series. Clin Infect Dis2004;39: 1214–1222.

69 Initiative to decrease the cv riskand increase the quality of care for patients living with HIV/AIDS executive summary circulation 2008.American heart association.

70. Riddler SA, Smit E, Cole SR, et al. Impact of HIV infection and HAART on serum lipids in men. JAMA 2003; 289:2978–2982.

71. Grunfeld C, Kotler DP, Shigenaga JK, et al. Circulating interferon-alpha levels and hypertriglyceridemia in the acquired immunodeficiency syndrome. Am J Med 1991; 90:154–162.

72. Sullivan AK, Nelson MR. Marked hyperlipidaemia on ritonavir therapy. AIDS 1997; 11:938–939.

73. Carr A, Samaras K, Thorisdottir A, Kaufmann GR, Chisholm DJ, Cooper DA. Diagnosis, prediction, and natural course of HIV-1 protease-inhibitor-associated lipodystrophy, hyperlipidaemia, and diabetes mellitus: a cohort study. Lancet 1999; 353: 2093–2099.

74. Segerer S, Bogner JR, Walli R, Loch O, Goebel FD. Hyperlipidemia under treatment with proteinase inhibitors. Infection 1999; 27:77–81

75. Van Leth F, Phanuphak P, Stroes E, et al. Nevirapine and efavirenzelicit different changes in lipid profiles in antiretroviral-therapy-naivepatients infected with HIV-1. PLoS Med 2004; 1:e19.

76. Gallant JE, Staszewski S, Pozniak AL, et al. Efficacy and safety of tenofovir DF vsstavudine in combination therapy in antiretroviral-naive patients: a 3-year randomized trial. JAMA 2004; 292:191–201.

77. Kumar P, Rodriguez-French A, Thompson M. Prospective Study of Hyperlipidemia in ART-naive Subjects taking Combivir/Abacavir(COM/ABC), COM/ Nelfinavir (NFV), or Stavudine (d4t)/Lamivudine(3TC)/ NFV (ESS40002) [abstract no. 33]. 9th Conference onRetroviruses and Opportunistic Infections. Seattle, WA, February24–28, 2002..

78. Dube MP, Stein JH, Aberg JA, et al. Guidelines for the evaluation andmanagement of dyslipidemia in human immunodeficiency virus(HIV)-infected adults receiving antiretroviral therapy: recommendations of the HIV Medical Association of the Infectious Disease Societyof America and the Adult AIDS Clinical Trials Group. Clin Infect Dis2003; 37:613–627

79. Mbulaiteye SM, Parkin DM, Rabkin CS. Epidemiology of AIDS-related malignancies: an international perspective. HematolOncolClin North Am 2003;17:673–696.

80. SteigmanCK, Anderson DW, Macher AM, Sennesh JD, Virmani R. Fatal cardiac tamponade in acquired immunodeficiency syndrome with epicardial Kaposi's sarcoma. Am Heart J 1988;116: 1105–1107.

81. O'ConnorPG, Scadden DT. AIDS oncology. Infect Dis Clin North Am 2000;14:945–965, vii.

PROFORMA

NAME:

AGE:

SEX:

ART NO:

COMPLAINTS:

Sob: present/absent duration: grade:

Chest pain:

Palpitation:

Pedal edema:

Abdominal distension:

Urine output adequate /decreased

PERSONAL HISTORY:

Smoker yes/no Alcohol yes/no

DM yes/no SHT yes/no

CLINICAL FINDINGS:

Pallor:

Pedal edema:

JVP

Pulse

BP

CVS

RS

INVESTIGATIONS:

CD4 COUNT:

Bd sugar

Urea

Creatine

LFT

CHG

ECG

ECHO

DURATION SINCE DIAGNOSIS

TREATMENT DETAILS

S. NO	AGE	SEX	STAGE	CD4	SYMP-TOM	SHT	DM	ALCOHOL	SMOKING	FBS	UREA	Sr.Cr	T.B	SGOT	SGPT	SAP	T.PRO
1	45	M	3	212	N	N	N	Y	Y	86	34	1	1	44	42	98	5.2
2	46	M	4	220	Y	N	N	Y	Y	87	32	.8	1.2	48	54	102	6
3	51	M	4	154	Y	N	N	N	Y	96	22	1.1	.8	54	56	86	5.8
4	56	M	3	112	Y	N	N	N	N	110	21	1.2	.8	47	42	88	5.4
5	38	M	3	112	N	N	N	Y	Y	115	16	1.1	1	55	54	104	5.4
6	56	M	3	143	N	N	N	Y	N	87	18	1	1	42	56	112	5.2
7	43	M	4	134	N	N	N	N	Y	94	23	.9	1.2	46	44	96	6.2
8	46	M	4	120	Y	N	N	Y	Y	68	32	.8	1.2	44	62	88	6.4
9	48	F	4	116	N	N	N	N	N	76	43	.9	1.2	48	68	86	6.2
10	43	M	3	126	Y	N	N	Y	Y	89	23	1	1.2	64	48	86	6.6
11	48	M	4	176	Y	N	N	Y	Y	75	22	1.	1.3	48	64	94	5.8
12	51	M	4	198	N	N	N	Y	N	78	23	1.3	1	68	48	98	5.8
13	47	F	3	154	N	N	N	N	N	93	32	1.2	.8	62	44	98	6
14	43	M	3	145	Y	N	N	Y	Y	95	34	1.6	.8	44	46	86	6
15	57	M	3	144	N	N	N	Y	Y	73	22	1	.8	56	42	84	5.5
16	43	F	3	135	N	N	N	N	N	85	21	1.8	1	54	55	82	6
17	46	M	3	246	N	N	N	Y	Y	86	20	1.4	1	42	47	88	6.2
18	51	M	3	235	Y	N	N	N	N	96	24	1	.8	56	54	112	6.2
19	34	M	3	136	Y	N	N	Y	Y	97	26	.8	1.2	54	48	107	6.4
20	56	F	3	160	N	Y	N	N	N	86	22	.8	1.2	42	44	82	5.8
21	56	F	3	220	N	N	N	N	N	86	32	.8	1.2	48	47	88	5.8
22	34	M	2	302	N	N	N	Y	Y	97	33	.8	1.2	64	55	86	6.2
23	51	M	3	312	N	N	N	N	N	96	42	1	.8	48	42	86	6.2
24	46	M	4	60	N	N	N	Y	Y	86	51	1.2	.8	44	46	94	5.8
25	43	M	4	98	Y	N	N	Y	Y	95	22	1.4	1.1	46	44	98	5.6
26	63	M	3	210	N	N	N	N	Y	88	54	.8	1.2	62	68	88	5.5
27	58	M	4	240	N	N	N	Y	Y	85	43	.8	1.2	68	48	118	6
28	53	M	3	108	N	N	N	N	Y	96	25	1	1.4	44	62	86	5.8
29	62	M	3	110	N	N	N	Y	Y	140	18	1.2	.8	68	44	86	5.8
30	59	M	4	82	N	N	N	Y	Y	132	22	1.2	1	48	66	102	5

S.NO	HB	TC	ESR	PLT	ECG FINDINGS	ECHO	ART
1	6	2300	32	120000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
2	7	4600	44	88000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
3	6	4400	46	98000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
4	9	3600	56	120000	SR,P PULMONALE,RT AXIS DEVIATION	RA RV DILATED,SEVERE PHT	N
5	9	2000	55	140000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
6	8	2200	46	55000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
7	8	4200	33	188000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
8	7	5000	65	36000	ST, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
9	5.4	4400	45	120000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
10	5.5	4600	45	88000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
11	6.4	5200	65	98000	SR ,LOW VOLTAGE COMPLEXES	MODERATE PERICARDIAL EFFUSION	N
12	7	5400	32	120000	SR,LOW VOLTAGE COMPLEXES	MINIMAL PERICARDIAL AND PLEURAL EFFUSION	Y
13	7.2	4800	34	100000	ST, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
14	9	4600	28	140000	ST, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
15	8.8	4600	33	120000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
16	7.6	4200	45	220000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
17	7	4200	42	120000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
18	8.4	3600	51	130000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
19	8.8	2600	33	98000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
20	8.6	4100	32	96000	SR LVH	N STUDY	Y
21	9	4200	45	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
22	6	4200	45	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
23	9	3600	65	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
24	9	2600	32	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
25	8	4100	34	220000	POOR PROGRESSION OF R WAVES	GLOBAL HYPOKINESIA DILATED CARDIOMYOPATHY EF 18%	Y
26	5.3	5000	28	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
27	4	4200	34	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
28	7.3	4400	65	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
29	8	8800	32	54000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
30	8.6	6800	54	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y

S.NO	AGE	SEX	STAGE	CD4	SYMP-TOM	SHT	DM	ALCOHOL	SMO-KING	FBS	UREA	SrCr	T.B	SGOT	SGPT	SAP	T.PRO
31	44	M	4	212	N	N	N	Y	Y	86	24	1	1.2	44	46	82	5.6
32	52	M	3	200	N	Y	Y	Y	Y	95	53	.8	1.2	46	42	88	6
33	42	F	4	156	N	N	N	N	N	85	43	.8	1.4	42	55	112	6.2
34	54	M	3	154	Y	N	N	Y	Y	88	32	1.2	.8	55	47	107	6.6
35	48	M	3	143	N	N	N	N	N	96	20	1	1	47	54	82	5.8
36	56	M	4	132	Y	N	N	Y	Y	98	18	1	.8	47	48	98	5.8
37	32	M	4	123	N	N	Y	N	Y	110	19	1.6	.8	46	44	102	6
38	44	F	2	210	Y	N	N	N	N	114	19	1.7	1.2	44	68	118	6
39	44	M	4	80	N	N	N	Y	N	63	18	.8	1.2	48	62	88	5.5
40	58	M	4	178	N	N	N	Y	N	86	20	.8	1.2	64	64	86	5.8
41	36	M	2	302	N	N	N	Y	Y	95	32	.8	1.2	48	42	86	5.8
42	44	M	4	98	N	N	N	Y	Y	85	43	1	1.4	68	60	94	6
43	49	F	3	102	N	N	N	N	N	88	54	1.2	.8	62	62	98	6
44	50	M	3	117	N	N	N	N	N	96	25	1.4	1	44	44	82	5.5
45	52	F	4	78	N	N	N	N	N	98	18	1.2	.8	68	68	88	6
46	61	M	4	98	N	N	N	Y	N	110	22	1.2	.8	48	48	112	6.2
47	43	M	3	100	N	N	N	Y	Y	114	34	1.4	1.2	64	64	107	6.2
48	52	M	3	212	Y	Y	N	Y	Y	63	54	2	1.2	48	48	98	5.4
49	51	M	4	204	Y	N	N	Y	Y	98	56	1.8	.8	54	42	82	5.2
50	44	M	3	210	N	N	N	Y	Y	96	71	2.1	.8	52	52	88	5.2
51	44	M	3	165	Y	N	N	Y	Y	98	18	1.6	1.2	55	47	102	6
52	52	F	4	168	N	N	N	N	N	110	19	1.7	1	47	54	118	5.5
53	38	F	4	210	N	N	N	N	N	114	C	.8	1	47	48	88	5.8
54	46	M	4	214	Y	N	N	Y	N	63	18	.8	1.6	46	44	86	5.8
55	44	M	4	306	N	N	N	Y	Y	86	20	.8	1.7	44	68	86	6

S.NO	HB	28TC	ESR	PLT	ECG FINDINGS	ECHO	ART
31	8	3100	32	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
32	8.2	5300	34	80000	NSR Q WAVES IN LEADS 2 3 AND AVF.	HYPOKINESIA OF INFERIOR WALL	Y
33	8	6800	28	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
34	7.2	4300	33	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
35	8	2000	28	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
36	8	2200	34	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
37	4	4200	45	50000	NSR LVH NO ST T CHANGES	GR 1 DIASTOLIC DYSFUNCTION	Y
38	5.3	5000	65	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
39	9	4400	32	120000	ST POOR PROGRESSION OF R WAVES	GLOBAL HYPOKINESIA DCM EF 26%	Y
40	8	6800	34	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
41	8	5500	28	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
42	7	4200	65	140000	NSR NQRS COMPLEX NO ST T CHANGES	MEDIASTINAL LYMPHADENOPATHY	Y
43	7	4200	46	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
44	8	4200	32	220000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
45	8	3600	8	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
46	9	2600	33	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
47	7.2	4100	46	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
48	8	6200	32	80000	NSR LVH NO ST T CHANGES	CON LVH	Y
49	8	3100	8	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
50	9	5500	42	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
51	7	6800	34	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
52	7	5500	28	50000	SR ,LOW VOLTAGE COMPLEXES	MODERATE PERICARDIAL EFFUSION	Y
53	8	4200	65	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
54	8	4200	46	120000	ST POOR PROGRESSION OF R WAVES NO ST T CHANGES	GLOBAL HYPOKINESIA DILATED CARDIOMYOPATHY EF 20%	Y
55	9	4200	32	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y

S.NO	AGE	SEX	STAGE	CD4	SYMP-TOM	SHT	DM	ALCOHOL	SMOKING	FBS	UREA	SrCr	T.B	SGOT	SGPT	SAP	T.PRO
56	64	M	3	260	N	N	N	Y	Y	110	19	1.2	.8	62	62	98	5.5
57	46	F	4	210	N	N	N	N	N	114	19	1.4	.8	44	44	82	6
58	44	M	4	110	Y	N	N	N	N	63	18	1.2	1	44	46	88	6.2
59	52	F	2	300	N	N	N	N	N	86	20	1.2	.8	48	44	88	6
60	55	M	3	230	Y	N	Y	Y	Y	95	32	1.4	1	64	48	112	6.2
61	44	F	3	210	N	N	N	N	N	85	43	2	.8	48	64	107	6.6
62	47	F	4	31	N	N	N	N	N	88	54	1.8	.8	68	48	82	5.8
63	52	M	3	212	Y	N	N	Y	Y	96	25	1.7	1.2	62	68	98	5.8
64	41	M	3	90	N	N	N	Y	Y	96	43	1.6	1	44	62	102	6
65	38	M	3	100	N	N	N	N	N	98	54	1.7	1	68	44	118	6
66	46	M	3	120	N	N	N	Y	Y	110	77	.8	.8	48	64	88	5.5
67	52	F	3	220	N	Y	Y	N	N	114	63	.8	1.2	64	48	86	5.8
68	43	M	3	250	N	N	N	Y	Y	63	19	.8	1.2	48	68	102	6
69	55	M	4	160	N	Y	N	Y	Y	86	19	1	1.2	68	62	118	5.5
70	43	M	4	200	N	N	N	N	N	95	18	1.2	1.2	62	44	88	5.8
71	47	M	4	80	N	N	N	Y	Y	85	20	1.4	1.4	44	44	86	5.8
72	45	M	3	108	N	N	N	Y	Y	88	32	1.2	.8	68	48	86	6
73	44	M	2	450	N	N	Y	Y	Y	96	62	1.2	1	48	64	94	6
74	39	M	4	108	N	N	N	Y	Y	145	34	1.4	2	66	44	98	5.5
75	40	M	4	100	Y	N	N	Y	Y	167	53	1	1.8	54	32	82	6
76	44	F	3	210	N	N	N	N	N	110	77	1.8	.8	64	48	102	6
77	46	M	4	98	N	N	N	Y	Y	114	63	1.7	1.2	48	68	118	6
78	54	M	3	342	N	N	N	N	N	63	19	1.6	1	68	62	88	5.5
79	57	M	4	210	N	N	N	Y	Y	86	19	1.7	1	62	44	86	5.8
80	60	M	3	246	N	Y	Y	Y	Y	95	18	.8	.8	44	44	102	6

S.NO	HB	TC	ESR	PLT	ECG FINDINGS	ECHO	ART
56	9	6800	33	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
57	8	4300	46	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
58	8.2	2000	32	140000	SR POOR PROGRESSION OF R WAVE	GLOBAL HYPOKINESIA DCM EF 20%	N
59	8	2200	33	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
60	7.2	4200	28	120000	NSR POOR PROGRESSION OF R WAVES	HYPOKINESIA OF LV APEX AND IVS EF 40%	Y
61	8	5000	34	140000	NSR RBBB COMPLEX NO ST T CHANGES	N STUDY	Y
62	8	4400	45	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
63	4	8800	65	188000	SR ,LOW VOLTAGE COMPLEXES	MODERATE PERICARDIAL EFFUSION	Y
64	5.3	4200	32	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
65	7.3	4200	34	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
66	7	3600	28	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
67	7	2600	65	140000	SR LVH WITH STRAIN PATTERN	AORTIC SCLEROSIS, CON LVH	Y
68	8	4100	34	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
69	8	6200	28	50000	NSR LBBB LVH NO ST T CHANGES	CON LVH	Y
70	9	3100	65	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
71	7.2	5500	46	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
72	8	6800	32	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
73	8	4300	8	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
74	10	2000	33	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
75	12	2200	46	120000	ST RT AXIS DEVIATION RBBB P PULMONALE RVH NO ST T CHANGES	RA RV DILATED PHT SEVERE	N
76	7.3	4200	45	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
77	7	3600	65	188000	NSR NQRS COMPLEX NO ST T CHANGES	MEDIASTINAL MASS.?LYMPH NODES	N
78	7	2600	32	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
79	8	4100	34	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
80	8	6200	28	120000	NSR LBBB NO ST T CHANGES	GRADE 1 DIASTOLIC DYSFUNCTION	Y

S.NO	AGE	SEX	STAGE	CD4	SYMPTOM	SHT	DM	ALCOHOL	SMOKING	FBS	UREA	SrCr	T.B	SGOT	SGPT	SAP	T.PRO
81	44	M	3	122	N	N	N	Y	Y	85	54	.8	1.2	66	44	88	5.5
82	46	F	3	240	N	N	N	N	N	86	25	.8	1.2	54	44	88	5.8
83	56	M	4	220	Y	N	N	Y	N	95	20	1	1.8	62	48	112	6
84	53	M	3	168	Y	Y	N	N	N	85	32	1.2	1.7	44	64	107	5.5
85	43	M	3	190	N	N	N	N	N	102	43	.8	1.6	46	48	82	5.8
86	42	M	4	200	N	N	N	Y	Y	132	54	1.2	1.7	44	68	98	5.8
87	32	M	4	100	N	N	N	Y	Y	99	25	1	.8	48	62	88	6.2
88	54	M	4	98	N	N	N	Y	Y	86	43	1	.8	64	46	82	6
89	52	M	4	110	Y	N	N	Y	N	60	18	.8	.8	66	44	98	6
90	36	M	3	122	Y	N	N	N	Y	110	20	.8	1	54	48	88	5.5
91	43	M	3	110	N	N	N	Y	Y	85	25	1.2	1.7	44	44	88	6.2
92	44	M	3	212	N	N	N	Y	Y	88	20	1	1.6	46	44	88	6.6
93	45	M	3	241	N	N	N	Y	Y	78	32	1	1	48	62	98	6.2
94	61	M	3	216	Y	Y	Y	Y	Y	108	85	44	1	64	44	82	6
95	34	M	4	30	N	N	N	Y	Y	109	86	43	1.2	66	46	88	6.2
96	54	M	4	120	N	N	N	Y	Y	99	51	.8	.8	54	44	88	6.6
97	43	M	3	110	N	N	N	N	N	110	45	.8	1.2	48	48	112	5.8
98	36	M	4	154	N	N	N	Y	Y	121	34	.8	1.2	64	64	107	5.8
99	32	M	3	208	N	N	N	Y	Y	132	24	1	1.2	48	44	82	6
100	45	M	3	186	N	N	N	Y	Y	154	32	1.2	1.2	68	42	76	5
101	44	M	3	110	N	N	N	Y	Y	89	32	.8	.8	64	47	102	6.2
102	46	M	4	123	N	N	N	N	N	75	43	.9	1	48	55	86	6.6
103	51	F	4	324	N	N	N	N	N	78	23	1	1	44	42	88	5.8
104	50	F	3	108	Y	N	N	N	N	93	22	1.	1.2	46	46	104	5.8
105	48	M	3	210	N	N	N	Y	Y	95	23	1.3	1.2	42	44	112	6

S.NO	HB	TC	ESR	PLT	ECG FINDINGS	ECHO	ART
81	8	2000	46	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
82	8	2200	33	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
83	9	4200	46	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
84	7.2	5000	32	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
85	8	2200	33	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
86	9	4200	28	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
87	8	5000	34	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
88	8.2	2200	45	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
89	8	4200	65	140000	NSR NQRS COMPLEX NO ST T CHANGES	MILD PERICARDIAL EFFUSION	N
90	7.2	6600	22	100000	NSR LAE N QRS COMPLEX NO ST T CHANGES	RHEUMATIC MS MVO 1.2 CM ^{2} MODERATE PHT	Y
91	8.2	2000	8	78000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
92	8	2200	22	100000	NSR RBBB NO ST T CHANGES	N STUDY	Y
93	7	4200	45	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
94	7	3600	65	76000	ST N QRS COMPLEX QS COMPLEX IN V1-V4	HYPOKINESIA OF ENTIRE ANTERIOR WALL EF 34%	Y
95	8	2600	32	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
96	8	4100	34	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
97	9	6200	28	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
98	7.2	3100	65	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
99	8	5500	34	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
100	9	6800	28	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
101	5.4	5000	46	140000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
102	5.5	4400	56	55000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
103	6.4	4600	55	188000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
104	7	5200	46	36000	ST, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
105	7.2	5400	33	120000	NSR NQRS COMPLEX OCC PREMATURE VENTRICULAR COMPLEX POOR PROGRESSION OF R WAVES NO ST T CHANGES	HYPOKINESIA OF ANTERIOR WALL EF 18%	Y

S.NO	AGE	SEX	STAGE	CD4	SYMPTOM	SHT	DM	ALCOHOL	SMOKING	FBS	UREA	SrCr	T.B	SGOT	SGPT	SAP	T.PRO
106	43	F	2	260	N	N	N	N	N	88	53	1.2	1.2	55	64	88	6.2
107	57	M	4	110	N	N	N	Y	Y	85	24	1.2	.9	42	48	82	6
108	47	M	3	280	N	N	N	N	N	96	43	1.4	1	47	48	112	6.6
109	51	M	3	310	N	N	N	Y	Y	98	32	.8	1	54	68	107	5.8
110	48	M	4	210	Y	N	N	Y	Y	110	20	1	1	48	62	82	5.8
111	46	M	3	100	Y	N	N	N	N	114	18	.8	1.2	44	44	98	6
112	43	F	3	145	Y	N	N	N	N	63	19	.8	1.2	68	68	102	6
113	56	M	4	243	Y	N	N	Y	Y	76	22	1.2	1	62	48	118	5.5
114	38	M	4	220	N	N	N	N	N	75	22	1.2	.8	44	64	94	6
115	56	M	3	240	Y	N	N	Y	Y	83	42	1	.8	56	48	96	6.2
116	51	M	4	110	N	N	Y	Y	Y	92	34	1.2	1.2	54	44	96	6.2
117	46	M	4	62	N	N	N	Y	Y	100	21	1	1.2	42	46	96	5.4
118	45	M	3	110	N	N	N	N	Y	73	18	.8	1.1	56	42	88	5.2
119	48	M	3	240	N	N	N	Y	N	84	18	.8	1.2	54	55	86	6.2
120	53	M	3	322	N	Y	N	Y	Y	86	20	.8	.8	42	47	102	6.4
121	43	M	3	220	N	N	N	Y	Y	95	32	1	.8	48	62	94	6
122	50	M	3	168	N	N	N	Y	Y	85	43	1.2	.8	64	64	98	5.5
123	49	F	3	779	Y	N	N	N	N	88	54	1.4	.8	48	42	82	6
124	34	M	2	340	N	N	N	Y	Y	96	77	1.2	1	68	60	88	6.2
125	35	M	4	150	N	N	N	Y	Y	98	63	1.2	.8	62	62	88	6
126	56	M	3	120	N	N	Y	Y	Y	110	19	1.4	1	44	44	112	6.2
127	53	M	4	110	Y	N	N	Y	Y	114	19	2	.8	44	46	107	6.6
128	42	M	3	144	N	N	N	Y	Y	63	18	1.8	.8	48	44	82	5.8
129	46	M	3	168	N	N	N	N	N	86	20	1.7	1.2	64	48	98	5.8
130	56	M	3	220	N	N	N	N	N	95	32	.8	1.2	48	64	102	6

S.NO	HB	TC	ESR	PLT	ECG FINDINGS	ECHO	ART
106	7	3100	33	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
107	8	6200	28	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
108	7	5500	28	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
109	8	6800	34	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
110	8	4300	32	120000	POOR PROGRESSION OF R WAVES	GLOBAL HYPOKINESIA DILATED CARDIOMYOPATHY EF 26%	Y
111	9	2000	66	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
112	7.4	2200	65	55000	NSR RVH RT AXIS DEVIATION	RA RV DILATED SEVERE PHT	Y
113	9.2	4200	46	188000	NSR LVH NO ST T CHANGES	ANTERIOR MITRAL LEAFLET PROLAPSE	Y
114	5.6	5000	32	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
115	8	4400	8	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
116	8.2	4600	33	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
117	8	5200	32	88000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
118	7.2	5400	44	86000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
119	8	4800	42	90000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
120	8	3300	42	100000	NSR LVH NO ST T CHANGES	CON LVH	Y
121	7.2	3600	8	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
122	8	2600	33	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
123	8	4100	46	120000	ST S1 Q3 T3 PATTERN RAE RBBB NO ST T CHANGE	RA RV DILATED WITH THROMBUS IN MAIN PULMONARY TRUNK	Y
124	9	6200	32	140000	NSR NQRS COMPLEXES NO ST T CNANGES	ANT MITRAL LEAFLET PROLAPSE	N
125	8	3100	33	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
126	8.2	5500	28	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
127	8	6800	34	140000	ST POOR PROGRESSION OF R WAVE	GLOBAL HYPOKINESIA DCM EF 34%	Y
128	7.2	4300	45	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
129	8	2000	65	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
130	8	2200	32	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y

S.NO	AGE	SEX	STAGE	CD4	SYMPTOM	SHT	DM	ALCOHOL	SMOKING	FBS	UREA	SrCr	T.B	SGOT	SGPT	SAP	T.PRO
131	36	F	2	340	N	N	N	N	N	86	32	.8	1.2	68	48	118	5.5
132	45	F	4	100	N	N	N	N	N	95	43	.8	1.2	48	64	88	5.8
133	46	M	4	118	Y	N	N	N	N	85	44	1	1.2	66	44	86	5.8
134	53	M	3	98	N	Y	Y	Y	Y	86	43	1.2	1.2	54	32	98	6.2
135	50	M	4	53	N	N	Y	Y	Y	95	54	.8	1.8	62	62	82	6
136	44	M	4	114	N	N	Y	Y	Y	85	25	1.2	1.7	44	44	88	6.2
137	41	M	4	88	Y	N	Y	Y	Y	88	20	1	1.6	46	44	88	6.6
138	37	M	3	198	N	N	N	Y	Y	96	32	1	1.7	44	48	112	5.8
139	48	M	3	186	N	N	N	N	N	96	43	.8	.8	48	64	107	5.8
140	46	F	3	144	N	N	N	N	N	98	54	1.2	.8	64	48	82	6
141	37	M	4	166	N	N	N	Y	Y	110	25	1.2	.8	66	68	98	6
142	39	F	3	158	N	N	N	N	N	114	43	1.2	1	54	62	88	5.5
143	44	M	4	36	N	N	N	Y	Y	63	54	1.8	1.2	62	44	86	6.2
144	43	M	3	206	Y	N	N	N	N	126	77	1.7	1.4	44	68	98	6.4
145	52	M	4	224	N	N	N	Y	Y	144	42	1.6	1	44	82	82	6
146	46	F	3	222	N	N	N	N	N	73	16	1.2	1.2	55	48	96	6
147	50	M	3	210	Y	Y	N	N	Y	85	18	1.6	1.2	47	64	88	5.5
148	38	M	3	176	N	N	N	Y	Y	86	23	1	1.3	54	48	102	6
149	44	M	4	268	N	N	Y	N	N	96	32	1.8	1	48	68	86	6.2
150	45	M	4	78	N	N	N	Y	Y	97	43	1.4	.8	44	62	88	6.2

S.NO	HB	TC	ESR	PLT	ECG FINDINGS	ECHO	ART
131	9	3100	65	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
132	7.2	5500	34	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	N
133	8	6800	28	50000	SR ,LOW VOLTAGE COMPLEXES	MODERATE PERICARDIAL EFFUSION	Y
134	9	6800	65	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
135	8	4300	46	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
136	8.2	2000	33	80000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
137	8	2200	46	120000	AF N QRS COMPLEX POOR PROGRESSION OF R WAVE	GLOBAL HYPOKINESIA DILATED CARDIOMYOPATHY EF 32%	Y
138	7.2	4200	32	140000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
139	8	5000	33	55000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
140	8	2200	28	188000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
141	4	4200	34	50000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
142	5.3	5000	45	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
143	10	4400	65	120000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
144	12	8800	22	140000	ST RT AXIS DEVIATION RBBB P PULMONALE RVH NO ST T CHANGES	RA RV DILATED PHT MODERATE	Y
145	7.8	4200	65	100000	NSR NQRS COMPLEX NO ST T CHANGES	N STUDY	Y
146	9	4800	65	88000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	N
147	8.8	4600	45	98000	NSR N QRS COMPLEX LVH NO ST T CHANGES	AORTIC SCLEROSIS	Y
148	7	4600	45	120000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y
149	7.2	4200	65	100000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	N
150	9	4200	32	140000	SR, N QRS COMPLEXES NO ST T CHANGES	N STUDY	Y

ABBREVIATIONS

HIV	-	Human Immunodeficiency Virus
ART	-	Anti Retroviral Therapy
PI	-	Protease Inhibitors
SHT	-	Systemic Hypertension
DM	-	Diabetes mellitus
AHA	-	American Heart Association
DCM	-	Dilated cardiomyopathy
EF	-	Ejection Fraction
PHT	-	Pulmonary Hypertension
PE	-	Pericardial Effusion
RA	-	Right atrium
RV	-	Right ventricle
LA	-	Left atrium
LV	-	Left ventricle
IVS	-	Interventricular septum
MS	-	Mitral stenosis
DD	-	Diastolic Dysfunction
NCEP	-	National Cholesterol Eradication Program
FBS	-	Fasting Blood Sugar
RFT	-	Renal function test
LFT	-	Liver function test
CHG	-	Complete haemogram
AACTG	-	Adults AIDS Clinical Trial Group

38

INSTITUTIONAL ETHICAL COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI-600 003

Telephone 25363970
Fax 044 2535115
Dated : 12.05.2010

L.Dis.No.14597/ME5/Ethics Dean/MMC/2010

Title of the work : "Cardiac Manifestations in HIV"

Principal Investigator : Dr. V. Nalini
Designation : PG in MD General Medicine
Department :
Madras Medical College & GGH, Ch-3.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 12th May 2010 at 2.p.m in Pharmacology Seminar Hall, Madras Medical College, Chennai -3

The members of the Committee, the Secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should get detailed informed consent from the patients/participants and maintain confidentiality.
2. You should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
3. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
4. You should not deviate form the area of the work for which you applied for ethical clearance.
5. You should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
6. You should abide to the rules and regulation of the institution(s).
7. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
8. You should submit the summary of the work to the ethical committee on completion of the work.
9. You should not claim funds from the Institution while doing the work or on completion.
10. You should understand that the members of IEC have the right to monitor the work with prior intimation.

SECRETARY
IEC, MMC,CHENNAI

CHAIRMAN
IEC,MMC, CHENNAI

DEAN
MADRAS MEDICAL COLLEGE,
CHENNAI -3